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The Journal of Laboratory and Clinical Medicine

VOL. IV. ST. LOUIS, JULY, 1919 No. 10

ORIGINAL ARTICLES

COMMUNICABLE DISEASE IN THE UNITED STATES ARMY DURING THE SUMMER AND AUTUMN OF 1918.*

BY COL. VICTOR C. VAUGHAN, M.C., U.S.A., AND CAPT. GEORGE T. PALMER, S.C., U.S.A., WASHINGTON, D. C.

PREFACE

THE following report on communicable disease in the army is based on the weekly telegraphic reports sent in to the Surgeon General from each camp, post, and army station, and upon the special reports made by division and camp surgeons, epidemiologists and others. The data are the most accurate obtainable at the time. We have observed a lack of agreement in reports from the same camp. The disagreements we have tried to fathom and eliminate and believe we have done so in the great majority of cases.

Before the complete truth about sickness in the army is known, it is necessary to analyze the records which eventually will reach the Surgeon General on the sick and wounded cards. Their compilation is a time-consuming task. In the meantime we are presenting this exposition of facts obtained from another angle, but whose interpretation we do not expect to see altered in any material degree by later analyses.

We desire to state that Major Alfred E. Shipley worked with us for nearly three months in the compilation of this report and we owe much to his counsel, advice, and help.

During a great part of the time this study has proceeded under the direction and with the kindly assistance of Lt. Col. George R. Callender.

*A report from the Section of Communicable Diseases, Division of Laboratories and Infectious Disease, prepared under the direction of Col. Frederick F. Russell, and published by permission of the Surgeon General. The writers are printing this as a preliminary report, subject to revision on the receipt of further data and will be grateful to all medical officers for more exact information on matters which came under their observation. Part II will be published in the August issue of this Journal.

We desire to thank division and camp surgeons, epidemiologists and other medical officers for the basic material of this report. Valuable data have been supplied by the United States Public Health Service, the Census Bureau, the Medical Bureau of the Navy, state and municipal health commissioners.

The tables and illustrations have largely been executed and owe their excellence to Hospital Sergeants Alvin D. Stultz and Edward H. Maitlen.

Section I

Introduction

The wise enactment and admirable execution of the draft law demonstrated that a great Democracy could mobilize, equip and place in the field, 3000 miles across the sea, an army efficient in battle. During the recent war our country prepared for service nearly four million men. They came, for the most part, not only willingly but eagerly, from farm, village and city. The university graduate and the illiterate, the poor and the rich, skilled and unskilled donned the khaki and began preparations for the task which they accomplished with speed and satisfaction. To care for the health of these men, to restrict the spread of disease among them, to restore so far as possible the sick and wounded to health has been the function of the medical department of the army. The task has fallen on us to tell to what extent and in what manner this work has been done.

Camps and cantonments for the reception and training of raw recruits came into being during the summer of 1917. The history of communicable disease in the army during the six winter months, October, 1917, to March, 1918, was prepared by the present authors, and published in the August, 1918, issue of the *Journal of Laboratory and Clinical Medicine* and reproduced in the September and October issues of the *Military Surgeon*. The purpose of the present contribution is to take up the account in April, 1918, and to carry it to the end of that year.

The procedure followed in the mobilization of our troops was not exactly that which would have been recommended by a board of sanitarians. This statement is made not with any thought of criticism. As hinted above, the draft law in itself, the execution of its provisions, and the results attained, will always be regarded as one of the greatest of our national achievements. The purpose of the mobilization was to assemble young civilians and to convert them into efficient soldiers in the shortest possible time, and was not to make a national demonstration in preventive medicine. In evaluating the results attained by the medical officer, the conditions under which he has been compelled to work must be duly considered. In this instance, both the soldiers and the medical officers, who were to have charge of their health, existed only in embryo, so far as military function is concerned when mobilization began. The hasty assembling of young men from every part of the country in groups of 27,000 or more brought into each camp practically every infection existent in the country among individuals of the same age group. The drafted men from each state were assembled at one or more points and held until a certain number had accumulated and then sent by troop trains to a designated camp. The

time covered in assembling and transferring varied from a few hours to as many days. It is reported by the camp surgeon at Wheeler that every troop train that reached that camp in the fall of 1917 brought from one to six cases of fully developed measles. In April, 1918, a colored draft contingent was assembled in Alabama and sent by troop train to Camp Custer, Michigan. On arrival, many cases of pneumonia were sent from this contingent directly to the base hospital at Custer. The coming of these troops not only increased the morbidity from pneumonia at Custer, but introduced a new type of the disease. This occurred frequently and at different camps. The control of epidemics under such conditions is a far different matter from that which concerns the local health authorities in a comparatively stable population.

For the convenience of our readers, we offer the following summary of the findings reached in our report on communicable disease during the winter of 1917-18:

1. Measles was nineteen times as prevalent in our training camps as in civilian life. The undue prevalence of this disease was especially noticeable in those camps which drew a large portion of their troops from rural populations. Organizations from the larger cities showed no more measles than appeared in the same age group in the cities from which they came. The number of cases in the different camps was determined by the number of susceptibles. Epidemics of this disease ceased when the susceptible material had been exhausted and reappeared when the spark and the inflammable substance were brought together. We fail to find convincing proof that any precautions taken limited the spread of this disease. Some of these measures may have retarded its spread, but none completely arrested it.

2. The pneumonias were twelve times as prevalent in the camps as in the civilian population. Again it was the rural and southern soldier who suffered most severely during the winter of 1917-18. However, in every camp the morbidity from this disease was higher than it was in the same age group at home. During the time covered by our report the morbidity from the pneumonias varied widely in the camps and these variations were apparently not influenced by the location of the camp but by the part of the country from which the troops came. Northern and urban men suffered least; southern and rural men most.

3. Meningitis was forty-five times more prevalent in the camps than in the civilian population. While no camp escaped this infection, it prevailed alarmingly in only three and these were camps which drew their troops from areas in which this disease was at the time endemic. These camps were Jackson, S. C., Beauregard, La., and Funston, Kan.

4. Scarlet fever was six times as prevalent in the camps as in civil life. Two camps, Beauregard, La., and Wheeler, Ga., had no cases of this disease. Pike, Ark., had the largest number and it may be well to remark that about one third of the troops in this camp were from the North. Apart from Pike, there was more scarlet fever during the winter months of 1917-18 among troops from the North and West than among those from the South. However, we are not justified in drawing any general conclusion from this fact.

5. Diphtheria was twice as prevalent in the camps as in civil life. Deaths

from this disease occurred in seven of the 29 camps whose records were studied. The annual death rates per 100,000 in these were as follows:

Pike	30
Funston	22
Dodge	18
Cody	17
Custer	10
Doniphan	8
Logan	7

The rate in the registration area in 1915 for all ages was 15.7.

6. Tuberculosis was thirteen times more prevalent among civilians than among soldiers. This is due to the elimination of the tuberculous in the examinations of the drafted men. That there were deaths from this disease during the first six months after mobilization indicates that some of the examiners were not skillful in the detection of this disease even in moderately advanced stages. At Camp Wheeler the annual death rate for this disease during the winter months of 1917-18 was 80 per 100,000, which is but slightly below that of the total population of Michigan without any examination or selection.

7. Typhoid fever and its allied diseases, including dysentery, were negligible, the annual death rate among all troops in the United States for the six winter months of 1917-18 being 1.3, while that for the state of Delaware for the same period was 55. It may be worthy of note that vaccination for this disease as practiced in the army gives protection against ordinary doses of infection, but the protective effects may be overcome by massive infection.

8. The following figures show the relative death rates from all causes in the army and in civilian life. The army rate is computed for the six winter months of 1917-18, while the civil rate is that of the corresponding months of 1915-16.

Place	Annual Death Rate per 1000
Army	9.1
U. S. Registration Area	
All Ages	14.3
Age 20-49	8.3
Age 20-39	6.8
Age 20-29	5.7

In the present article the time from April to December, 1918, is divided into two parts for statistical treatment. This is done in order to set forth the seasonal influence on disease. Ordinarily we should have grouped together the months from April to September inclusive, calling this the "summer season" as contrasted with the months from October to March inclusive, which was regarded as the "winter season." However, the occurrence of the influenza epidemic in September and the following months has caused us to alter this grouping and treat as the "summer season" the period from April to August inclusive. The four remaining months of 1918 are then treated under the term "autumn season."

The annual death rate per 1000 for the summer season was 5.7, practically the same as that of civilians in the age of group 20 to 29 years during the six winter months. For the corresponding five summer months of the year 1916, the death rate from all causes for this age group in civilian life is estimated to be 4.6. Consequently, the low rate of 5.7 of the army is still a trifle above

that of civilian life. This is a remarkable showing, for although the army is composed of young men selected on account of superior physical make-up and men who might be expected to have a lower death rate than the average among those of their own age, still, when we consider the hazard that is always associated with the mobilization of very large numbers for military service, the rate of 5.7 for the summer season may be pointed to with pardonable pride.

During the summer season pneumonia continued to be the chief cause of death. However, the greater part of this fatality occurred during the months of April and May. Contrary to the experience of the previous winter, the pneumonias prevailed more extensively in the middle western camps than elsewhere. In many camps measles was present but to a far less extent than during the winter period. The prevalence of this disease during this period was undoubtedly dependent upon the number of susceptibles in the camp. During the winter months, Wheeler stood third in measles incidence while in March there were no cases in this camp. During the winter, Wheeler had been so permeated with this disease that every susceptible individual was affected, and then the diseased ceased. There were no new men added to Wheeler until autumn, and susceptible material for the continuation of measles was lacking.

In discussing typhoid fever in our previous report, we stated that the low typhoid rate at that time might in part be attributed to the fact that the winter season was not the typhoid period of the year and the real test of preventive inoculation would come during the summer. It was the summer season which witnessed the typhoid scourge in 1898. Now that we have passed through the summer of 1918 and have experienced a typhoid death rate but little greater than that of the preceding winter, we can rest quite comfortably in the conclusion that the control of this and its allied diseases is within the power of man. The annual typhoid death rate per 100,000 during the summer season was 3.3, but these figures do not do justice to the preventive measures employed, for many of the cases that did occur were among men who had contracted the infection prior to receiving their vaccination.

Malaria, another warm season disease, increased during the summer to a slight degree only. For the most part, malarial admissions to the hospital were among men who had the infection prior to entering service. Although some of the camps were in malarial districts, there is no evidence that any number of infections originated in these camps.

Coming now to the "autumn season," counting September to December inclusive, we find ourselves facing a wholly different condition. During this period the unparalleled pandemic of influenza reached and spread to the remotest parts of this country. Among our soldiers the death rate rose from 5.7, that of the summer season, to 48.5 for the autumn season, an increase of nearly tenfold. Still more impressive is the actual number of deaths. During these four months there occurred in the various army camps in this country 23,712 deaths, no less than 94 per cent of which was due to influenza and complicating pneumonia. This means that 1.5 per cent of the military forces was effectually eliminated in the infancy of their career.

Out of nearly four million men who passed through the army camps of this country from September, 1917, to January, 1919, 30, 859 have died from disease

before reaching the port of embarkation. Up to November 26, 1918, 16,000 of our soldiers in France died of disease. This makes a total of 47,000 American soldiers who gave up their lives in our hospitals, serving their country less conspicuously, but none the less heroically, than those who died on the fields of battle. Of the two million men landed in France, 40,000 perished in combat according to Pershing's report dated Nov. 26, 1918. Of the total 87,000 deaths, 54 per cent was from disease. As war records go, this is not an unusual showing. Of the deaths in our Civil War among northern troops, 66 per cent was from disease; in the Franco-Prussian War, the French Army, 68 per cent, German Army 31 per cent; in the Spanish-American War, United States Army, 84 per cent; in the Russo-Japanese War, Japanese Army 32 per cent, Russian Army, 29 per cent. In the American Expeditionary Force the percentage dying from disease up to November 26, 1918, was 29.

Preventive medicine is making rapid progress. Malaria and typhoid fever, once the scourges of armies, are well in hand. Typhus is no longer an enigma, but still requires close watching. The pneumonias remain and constitute at present the greatest cause of death in all armies. Protective inoculation against the pneumonias has been tried only recently, and the results are promising. The combination of influenza and pneumonia has been most distressing and disastrous both in military and in civilian populations. During the autumn season of 1918, civilian communities suffered greatly, but on account of the high concentration of susceptible material in our camps, the death rate among soldiers has been higher than among civilians. No part of the world has escaped this great scourge. In the past, there have been pandemics, but none, so far as we have statistical evidence, has wrought such heavy destruction as this. Hitherto the pandemic of 1889-90 has been looked upon as the most widespread and probably the most fatal. At that time more than 40 per cent of the population of Massachusetts was affected, but the death rate was not so high. The pandemic of 1918, when compared with that of 1889-90 is estimated to have caused *six times as many deaths.*

During the four autumn months of 1918, 338,343 cases of influenza were reported to the Surgeon General. This means that in the camps of this country *one out of every four men had influenza.*

The combination between influenza and pneumonia during the fall of 1918 seems to have been closer and more destructive than in any previous pandemic. During the autumn season there were reported to the Surgeon General 61,691 cases of pneumonia. This means that *one out of every twenty-four men encamped in this country had pneumonia.*

During the same period 22,186 men were reported to have died from the combined effects of influenza and pneumonia. This means that among the troops in this country *one out of every sixty-seven died.*

This fatality has been unparalleled in recent times. The influenza epidemic of 1918 ranks well up with the epidemics famous in history. Epidemiologists have regarded the dissemination of cholera from the Broad Street Well in London as a catastrophe. The typhoid epidemic of Plymouth, Pa., of 1885, is another illustration of the damage that can be done by epidemic disease once let loose. Yet the accompanying table shows that the fatality from influenza and

pneumonia at Camp Sherman was greater than either of these. (See Table 1 and Chart I). Compared with epidemics for which we have fairly accurate statistics, the death rate at Camp Sherman in the fall of 1918 is surpassed only by that of plague in London in 1665 and that of yellow fever in Philadelphia in 1793.

The plague killed 14 per cent of London's population in seven months' time.

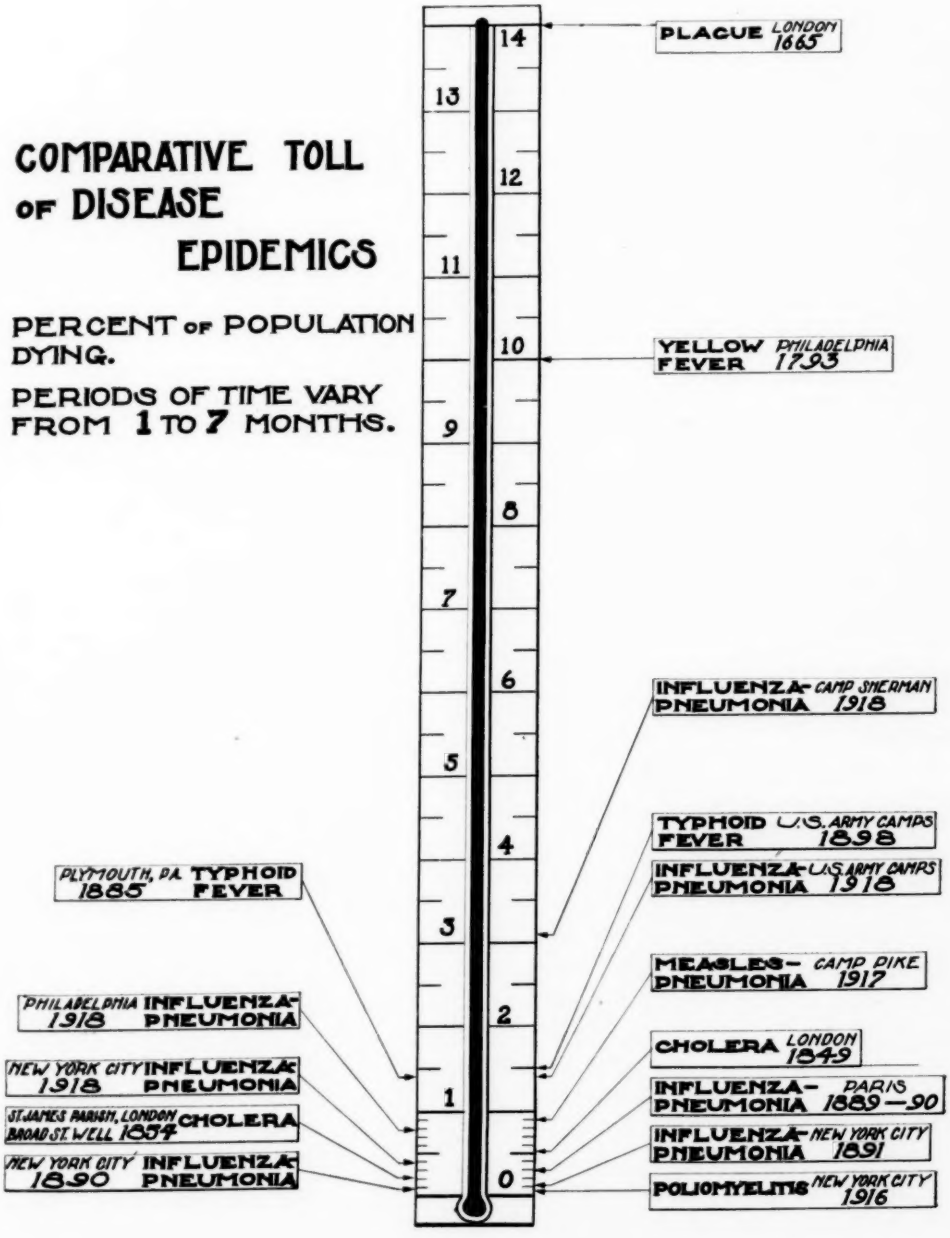


Chart I.

TABLE 1

COMPARATIVE TOLL OF DISEASE EPIDEMICS

	DISEASE	PLACE	YEAR	PERIOD	POPULATION	DEATHS DURING PERIOD	% POPULATION KILLED DURING PERIOD	% POPULATION KILLED PER MONTH
1	Plague	London	1665	7 months	500,000	68,593	14.	2.0
2	Yellow fever	Philadelphia	1793	4 months	40,100	4,041	10.	2.5
3	Influenza-pneumonia	Camp Sherman	1918	7 weeks	35,100	1,073	3.1	1.9
4	Typhoid fever	U. S. Army Camps	1898	4 months	108,000	1,580	1.5	.38
5	Influenza-pneumonia	U. S. Army Camps	1918	4 months	1,500,000	21,500	1.4	.35
6	Typhoid fever	Plymouth, Pa.	1885	1 month	8,000	114	1.4	1.4
7	Measles-pneumonia*	Camp Pike	1917	14 weeks	28,100	252	.90	.28
8	Influenza-pneumonia*	Philadelphia	1918	7 weeks	1,761,000	13,500	.77	.48
9	Cholera	London	1849	23 weeks	—	—	.51	.096
10	Influenza-pneumonia*	New York City	1918	7 weeks	5,740,000	14,700	.3	.24
11	Influenza-pneumonia*	Paris	1889-1890	7 weeks	2,260,000	6,500	.29	.18
12	Cholera	St. James Parish, London ("Broad St. Well")	1854	17 weeks	318,000	700	.22	.056
13	Influenza-pneumonia*	New York City	1891	1 month	1,681,000	1,818	.11	.11
14	Influenza-pneumonia*	New York City	1890	1 month	1,631,000	1,370	.08	.084
15	Poliomyelitis	New York City	1916	5 months	5,602,000	2,407	.043	.009

*Mortality computed indirectly from excess deaths from all causes above norm.

Yellow fever destroyed 10 per cent of the population of Philadelphia in four months. In seven weeks influenza and pneumonia killed 3.1 per cent of the strength at Camp Sherman. If we consider the time factor, these three instances are not unlike in their lethality. The plague killed 2 per cent of the population in a month, yellow fever 2.5 per cent and influenza and pneumonia 1.9 per cent.

In four months typhoid fever killed 1.5 per cent of the soldiers encamped in this country during the war with Spain. Influenza and pneumonia killed 1.4 per cent of the soldiers in our camps in 1918 and it also covered a period of four months.

During the winter of 1917-18, Camp Pike showed the highest death rate of the larger camps, this being due for the most part to pneumonia to some extent following measles. In fourteen weeks Pike lost 0.9 per cent of its strength. This is about one-third of Sherman's loss but the deaths at Sherman occurred in one-half the time. In the winter of 1917-18, Camp Beauregard showed the highest weekly incidence for measles. It amounted to an annual admission rate per 1000 of 2,700. In the fall of 1918 Beauregard had an influenza admission rate during one week of 15,000. The two epidemic periods at Beauregard have been

contrasted graphically in Chart II. The 1917 epidemic looks insignificant compared to that of 1918, and yet at the time it was regarded with grave concern. Philadelphia headed the large cities in influenza fatality in this country, losing 0.8 per cent of its population. This is about one-fourth of the loss at Sherman.

The pandemic of influenza in 1918 seems to have been more closely associated with the pneumonias than appears in any previous pandemic. From

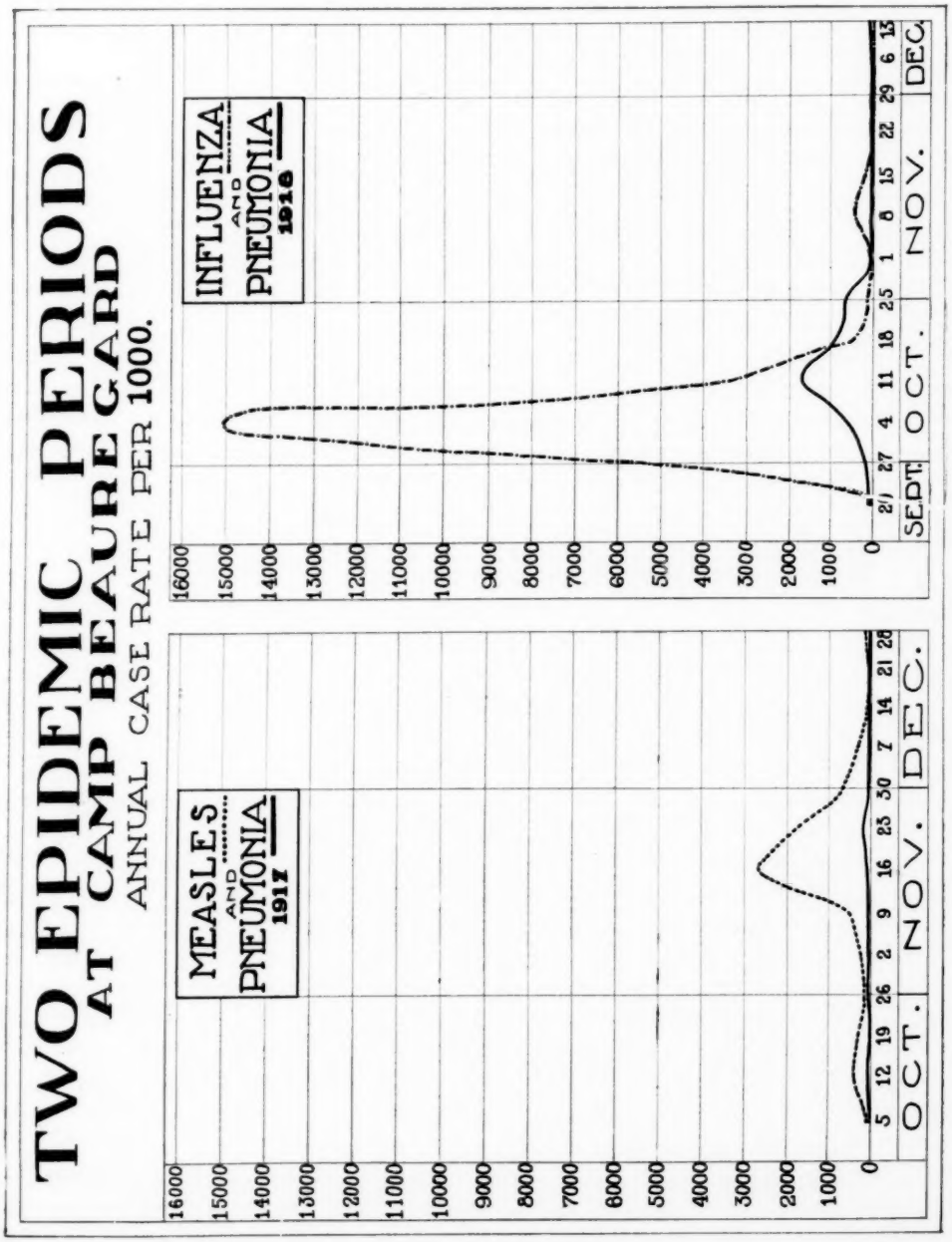


Chart II.

the reports as sent to the Surgeon General's Office, it appears that uncomplicated influenza was not by any means a fatal disease and that the high death rate was due to the pneumonias which followed. Pneumonia is a serious disease at all times. Recent records for the United States Army show that the case mortality rate for this disease has been as follows during the different periods of the last two years:

The year 1917	11.2%
6 winter months, 1917-18	23.1%
5 summer months, 1918	18.8%
4 autumn months, 1918 (influenza period)	34.4%

It is not strange that once pneumonia secured a foothold in patients already weakened by influenza their chances of recovery were lessened.

In 1893 a German bacteriologist, Pfeiffer, announced his discovery of the bacillus which has since borne his name and which he believed to be the specific cause of influenza. In this country Pfeiffer's claims have never been universally accepted. We will have more to say on this point from time to time as the evidence unfolds itself.

The pneumococcus has been long regarded as the chief cause of pneumonia. Of this organism four distinct types are recognized in this country. The fourth type is in reality a heterogeneous group which includes many organisms which may cause pneumonia and yet whose agglutinations and other reactions have not been recognized with a sufficient degree of accuracy to be accepted as a means of identification. During the past year, it has been impressed upon us more forcibly than before that other oganisms than the pneumococcus may cause clinical pneumonia. The streptococcus and the staphylococcus, at least certain varieties of these organisms, have produced clinical pneumonia in our camps. One reading the reports that have been sent in from army camps in all parts of the country is impressed by the lack of agreement as to the bacteriology which has been responsible both for influenza and the accompanying pneumonia. So far as influenza is concerned, the descriptions of the clinical symptoms agree. There is no question but that the same disease clinically has existed in Massachusetts, Kansas and California. In one place, however, the Pfeiffer bacillus, in another the streptococcus hemolyticus and in a third some form of the pneumococcus has been believed to be the cause of the disease. Suspicion has also been cast upon various strains of streptococcus, the micrococcus catarrhalis and the staphylococcus. In one laboratory there has been no difficulty in isolating the Pfeiffer bacillus from the throats of influenza patients; in another this organism has not been found and good reason is furnished to support the belief that the disease is of purely streptococcic origin. These differences of opinion with respect to the initial cause of the infection have existed to a like degree in the case of the pneumococcic organisms which have been recovered from the throats of the sick. One camp reports more of one type of pneumococcus than another. There is agreement merely in the excessive numbers of Type IV, but we must remember that this is only a "waste basket" group. Pneumococci which do not respond to reactions characteristic of Types I, II, or III are placed in group four. It follows, therefore, that the

information that type four has prevailed during the epidemic is not altogether satisfactory.

In the face of these contradictory reports, not only from army camps, but from civilian laboratories as well, we are handicapped in determining the true cause of the disease. Basing our opinion on the information which is given in detail in the pages to follow, we make the following tentative statement:

1. Influenza is an acute, highly infectious disease of unknown origin, characterized by the production of a marked leucopenia which results in withdrawal of the natural defenses of the body and the opening up of the paths of invasions for other pathogenic organisms which may be present.

2. We are of the opinion that one reason for the variation in the manifestations and course of the disease in different communities has been due to difference in the combinations of organisms which have worked symbiotically with the specific cause of influenza. This accounts for the finding of one organism prevalent in one place and another organism dominant in another.

3. We are of the opinion that the epidemic of influenza which occurred in the fall of 1918 was not a new entity but a recurrence or reappearance in a more virulent form of the disease which had prevailed in the various Army Camps during the previous year. Our justification for this statement is that there are many incidents of people who largely escaped the disease in the fall and who had experienced or lived through a similar but much milder epidemic during the previous spring. In other words, we may say the soldiers who had clinical influenza in camp prior to August and those who, although having no clinical manifestations of the disease, lived through such an epidemic, were less gravely affected when the more virulent organisms reached them in the fall.

4. We believe that the largest single factor influencing the spread of influenza is the susceptibility of the individuals among whom it has been introduced. If these individuals have been once attacked by the disease even in a mild form or lived through a mild epidemic without showing clinical symptoms, they suffer less when the disease is again introduced. Among communities not previously exposed to influenza, this disease has usually involved from 20 to 50 per cent of the personnel. The exact number affected is determined by the number of people who are naturally immune or have secured immunity by previous exposure.

5. It appears that natural immunity gives way before exposure, overwork and fatigue, as was demonstrated years ago by Pasteur in his experiments on birds with anthrax. Likewise, it is possible for human beings to have their resistance lowered by exposure to unaccustomed environment so that although naturally immune, the standard of immunity is reduced to the point where the influenza virus gains admittance, and overcomes the lowered resistance.

6. We believe that not only the rapidity with which the disease spreads, but its virulence is in direct proportion to the density of the susceptible population. In communities such as army camps and large cities, the contact of individuals is so close and so intimate that even though extra precautions are taken it is quite impossible to prevent disease from ultimately reaching all persons. Precautions such as quarantine, spreading out of the personnel, closing places of assembly, delay the progress of the disease but fail to wholly prevent it.

At present and with the data at hand we can not go further with reference to the causation and diffusion of the pandemic. In this paper it is our province to describe the spread of the disease through the army camps and to interpret the significance and conditions of its distribution, with the ultimate aim of suggesting measures which may be of value in the future in combat with the same disease.

Influenza and pneumonia so far surpassed other diseases during the autumn season that they overshadowed all others. It was expected that on account of seasonal change and the assembling of many susceptibles during the fall, measles would again become an immediate factor among the communicable diseases in our camps. In fact, there was no such outburst of this disease as occurred during the previous fall. Without exaggeration it may be said that for the time being, at least, influenza and pneumonia suppressed other infectious diseases. Typhoid fever increased to a barely noticeable degree. The death rate from this disease was somewhat higher, but the total number is so small as to barely warrant comment and not to justify any definite conclusion. Scarlet fever and malaria were both lower than during the summer. In fact there was but one scarlet fever outbreak of any importance and that occurred at Camp Hancock. Within two weeks over 300 cases were reported and this marks the largest scarlet fever epidemic that occurred in the camps in this country at any time. Meningitis increased although it did not reach the prevalence of the previous winter. The weekly incidence curve for all troops in this country, as will be shown in Section II, suggests that meningitis was in some instances a sequela to influenza. The greatest meningitis incidence corresponds with the influenza peak. Diphtheria showed no material increase. Deaths from tuberculosis were higher in the autumn than in the two previous periods, the death rate rising from 18 per 100,000 during the summer to 46 in the autumn. The rate for the previous winter was 15. The most plausible explanation for this increase in deaths is that dormant and incipient cases introduced into the army during the preceding year had accumulated and possibly were hastened into the acute stage both by the duties of camp life and by the prevalence of the epidemic of influenza and pneumonia. Quite naturally there had been from the time of the first assembling of troops an accumulation of tuberculous individuals, inasmuch as such men were not discharged but were kept in the army and under governmental control and provision.

Looking back over the years 1917 and 1918, we are deeply impressed with the influence of immunity in governing the spread of infectious diseases. Apparently measles is a very specific disease, one attack immunizing an individual for a lifetime, and there is no other process so far as we can see by which immunity to this disease may be secured. Introduce measles among a susceptible population to which it has been a stranger and it will burst forth and spread rapidly. This is what happened in the fall of 1917. On the other hand, meningitis is a disease to which a large portion of the population is naturally immune. It is a striking fact that in the army camp meningitis has not shown marked effects of contact infection. Where cases have occurred they have been for the most part, at least, sporadic and apparently without connection. This phenomenon has been observed throughout the seasons. As the rural population is

more susceptible to measles on account of lack of previous exposure, the same is true of pneumonia to a certain degree at least. In our camps it is the rural rather than the urban man who contracts pneumonia. The latter is immune largely not because he has had pneumonia, but because he has been brought frequently in contact with pneumonia causing organisms and this slight but repeated inoculation has given him a certain degree of immunity. Whereas the rural man is normally more susceptible to the pneumonias when brought in contact with the organisms causing these diseases, his susceptibility may be increased still further by such environmental factors as the weather and fatigue. The proof of this has been amply demonstrated in the numerous cases where organizations have been shifted from one camp to another. A journey by troop train has almost invariably resulted in wide inoculation and the development of a common salivary flora. Incidences of this kind will be in evidence as we develop camp histories. Men who were apparently well when starting out have been taken off the train sick and immediately transferred to the hospital. There are not isolated instances of this, but it is a common experience. The influence of travel and fatigue in lowering resistance is applicable to both urban and rural men, but is more noticeable in the latter because their susceptibility is greater to start with.

A general summary of the occurrence, dissemination and methods employed in the various camps to control disease will be given later. It is sufficient to say in this introductory paragraph that the diseases which have prevailed among our troops have been for the most part inevitable on account of our ignorance of their etiology and the methods necessary for their prevention. Along these lines there is a need for a vast amount of experimental work. We may hope that this will be done and information obtained which will enable us, should similar conditions arise in the future, to know how best to proceed to mobilize a great army and at the same time preserve the men from death by sickness. At present the most encouraging field in which to look for this knowledge is to be found in that of preventive inoculation. The success attained with smallpox, typhoid, diphtheria and tetanus points the way. It is to be hoped that the pneumonias will yield to like procedures. It remains for the bacteriologist to press vigorously his inquiries into the etiology and epidemiology of the respiratory diseases, especially the pneumonias, influenza, scarlet fever and measles. When the viruses of these diseases have been definitely identified and the avenues by which they are disseminated are discovered, it will be possible to still further greatly reduce the deaths from sickness not only in military but in civilian life.

Section II

Death and Disease in the Army as a Whole

A. Causes of Death

We have pointed out in our published review of disease in the army for the six winter months, October, 1917, to March, 1918, inclusive, that diseases of the respiratory tract caused the greatest number of deaths. Of the respiratory diseases pneumonia stands above all others. During the winter period in question, respiratory diseases, which were interpreted to cover meningitis, pneumonia,

measles, scarlet fever, diphtheria and tuberculosis, were responsible for 77 per cent of all deaths among troops quartered in the U. S. Pneumonia alone was responsible for 61½ per cent of all deaths. During the five summer months from April to August, 1918, inclusive, pneumonia was responisble for 49.3 per cent. During the autumn season, the four months from September to December, 1918, inclusive, pneumonia and influenza were responsible for 93.7 per cent. Pneumonia therefore stands out as the one infection against which the greatest efforts should be directed.

The actual number of deaths and the annual rate per 100,000 from each cause for the summer season are given in Table 2. The average strength of the troops in this country at this time was 1,420,000. This number is a fairly representative average, for at no time did the strength drop below 1,300,000, nor

TABLE 2

CAUSES OF DEATH
All Troops in the United States
Five Summer Months, Week Ending April 5, to Aug. 30, 1918
Average Strength—1,420,000

DISEASE	NUMBER OF DEATHS	ANNUAL RATE PER 100,000
Pneumonia	1679	279.
Diseases other than here mentioned*	665	111.
Meningitis	248	41.3
Tuberculosis	109	18.1
Empyema	86	14.3
Scarlet Fever	26	4.3
Typhoid and Paratyphoid fever	20	3.3
Measles	19	3.2
Diphtheria	5	.84
Total disease	2857	474.
Causes other than disease**	558	92.8
All causes	3415	567.

*Includes appendicitis, septicemia, heart disease, nephritis, etc.
**Includes burning, fracture, suicide, gunshot wounds, etc.
(See Chart III)

did it at any time exceed 1,700,000. The strength increased gradually from April through August, the maxium occurring in the week ending August 16, there being at that time 1,678,000 men on the army rolls in this country.

Taking up the summer season we see from Table 2 that next to pneumonia as a cause of death, is the group designated "diseases other than here mentioned," which includes for the most part the noncommunicable diseases, such as appendicitis, septicemia, heart disease, etc. The annual death rate per 100,000 for pneumonia was 279; that, for this miscellaneous group of many diseases, 111. Next to "causes other than disease," meningitis stands third on the list with a death rate of 41.3. This is followed by tuberculosis with a rate of 18.1, and empyema with a rate of 14.3. The last named disease was not listed separately in the fall of 1917 but increased in prominence, and as will be seen, is a factor of no little consequence. Next follows scarlet fever and trailing after this are typhoid, measles, and last of all diphtheria. We regard it as a remarkable fact that 1,420,000 men can live together in the close association necessary

in camp and cantonment without there resulting more than five deaths from diphtheria. It is a tribute to preventive medicine that this disease has been rendered so impotent.

There were but twenty deaths during this time from typhoid and paratyphoid, and these were for the most part among men new to the service who had received their inoculation after the typhoid bacillus had gained entrance to their bodies. The deaths from causes other than disease total 558, and place this third in the list with an annual rate per 100,000 of 92.8.

The deaths and rates for the autumn season are given in Table 3. The average strength for this period was 1,493,000, the maximum at any one time being for the week ending September 13, 1918, when there were listed 1,689,000 men. The lowest strength occurred in the week ending December 27, 1918,when demobilization was well under way, there being at that time 1,036,000 men. As in the summer and in the previous winter, pneumonia is the great reaper. This season marked the period of the influenza epidemic, during which 21,053 deaths are charged to pneumonia. Influenza was recorded as having caused 1,133 deaths. Although these figures are given separately in Table 3, we have combined the two in Chart III. The annual death rate per 100,000 from pneumonia was 4,300. This is nearly eight times the rate of the previous winter, which was considered at the time high. The group of diseases labeled "other than here mentioned" accounted for 512 deaths. This would mean an annual rate of 105. "Causes other than disease" were responsible for a rate of 80.2, and this stands fourth in importance when considering influenza separately from pneumonia. Typhoid, measles, scarlet fever, diphtheria, each had less than 22 deaths, there being five from diphtheria for the four months this season, which is the same number that occurred during the five months of the summer season.

TABLE 3

CAUSES OF DEATH

All Troops in the United States

Four Autumn Months, Week Ending September 6, to December 27, 1918
Average Strength—1,493,000

DISEASE	NUMBER OF DEATHS	ANNUAL RATE PER 100,000
Pneumonia	21,053	4300.
Influenza	1,133	232.
Diseases other than here mentioned*	512	105.
Meningitis	244	49.8
Tuberculosis	225	46.0
Empyema	102	20.8
Typhoid and paratyphoid fever	21	4.3
Measles	14	2.9
Scarlet fever	10	2.0
Diphtheria	5	1.0
Total disease	23,319	4770.
Causes other than disease**	393	80.2
All causes	23,712	4850.

*Includes appendicitis, septicemia, heart disease, nephritis, etc.
**Includes burning, fracture, suicide, gunshot wounds, etc.

(See Chart III)

To better appreciate the significance of the deaths from these various causes we have presented in graphic form (see Chart III) death rates for the three seasons, winter summer and autumn, placing together the rates for each disease. A casual survey of this chart indicates that as between the three seasons, pneumonia decreased 50 per cent in the summer, and then jumped enormously

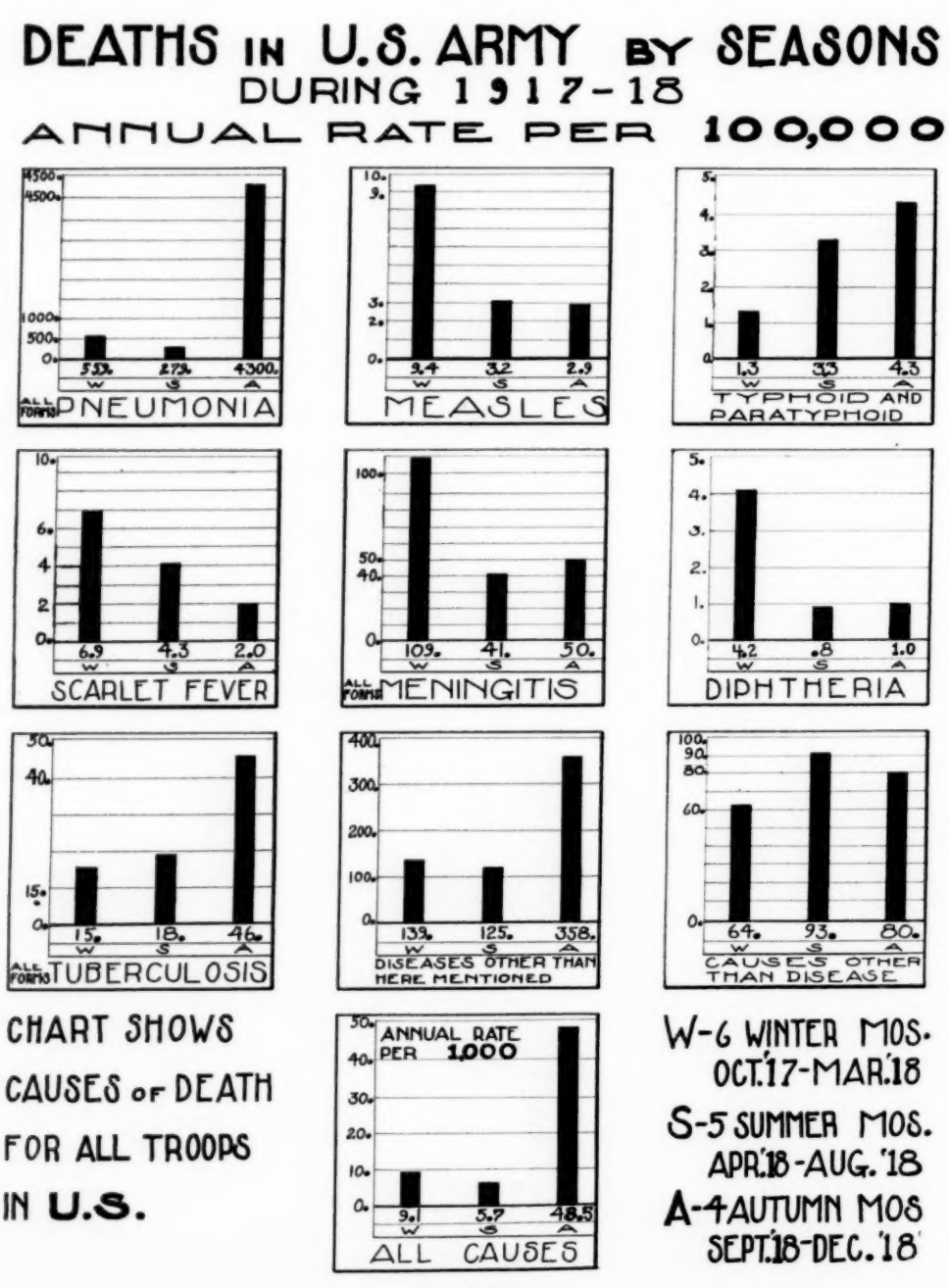


Chart III.

in the autumn. Measles in the summer time was one-third what it was the previous winter, and there was still a further drop in the autumn. The deaths from typhoid increased from winter to autumn. Scarlet fever decreased progressively. Meningitis was greatest during the winter season, a fourth this figure during the summer, and increased slightly in the autumn. Diphtheria fell off perceptibly during the summer and autumn. Tuberculosis increased slightly during the summer and perceptibly in the autumn. This increase may

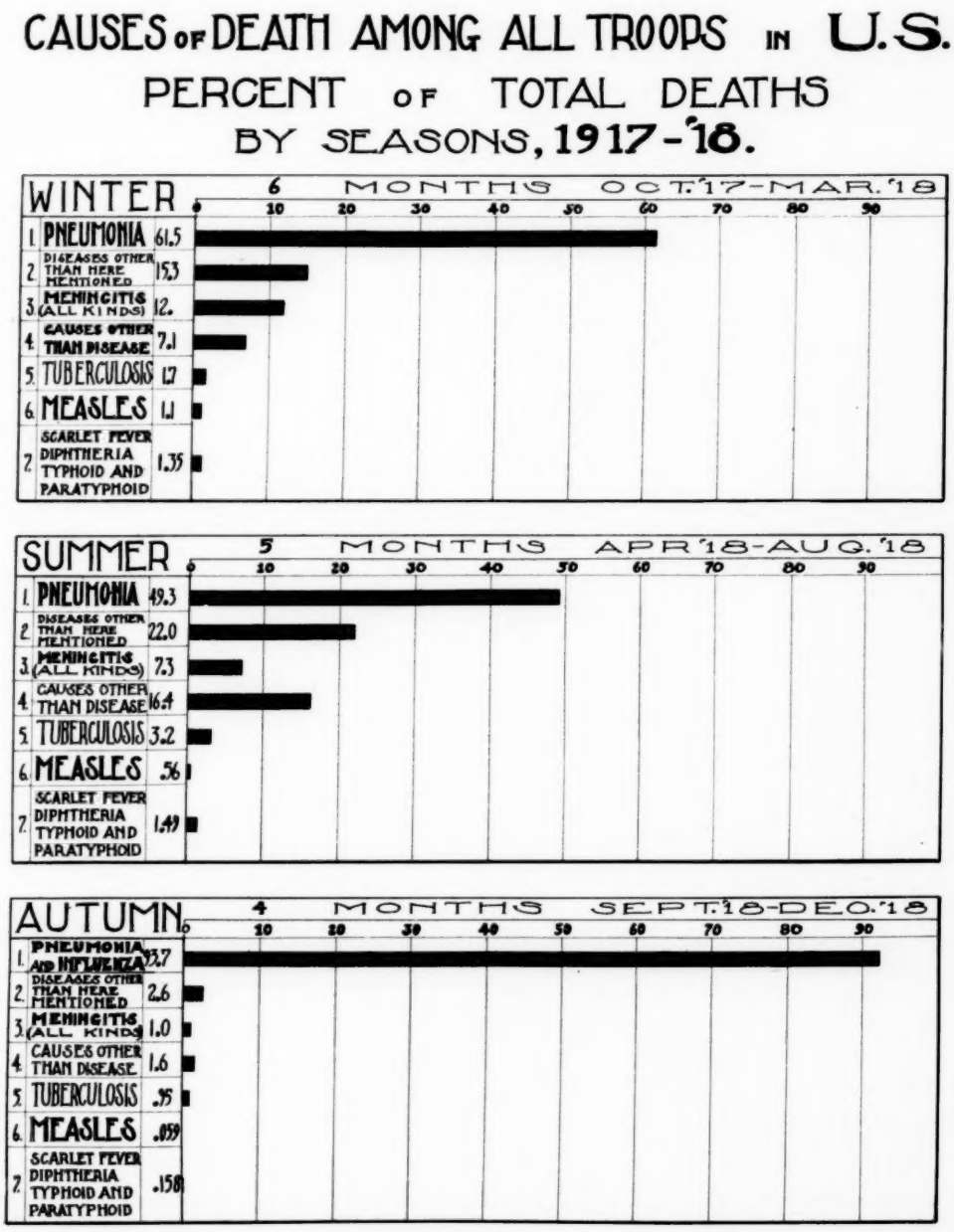

CAUSES OF DEATH AMONG ALL TROOPS IN U.S.
PERCENT OF TOTAL DEATHS
BY SEASONS, 1917-'18.
WINTER
6 MONTHS OCT.'17-MAR.'18
0 10 20 30 40 50 60 70 80 90
1. PNEUMONIA 61.5
2. DISEASES OTHER THAN HERE MENTIONED 15.3
3. MENINGITIS (ALL KINDS) 12.
4. CAUSES OTHER THAN DISEASE 7.1
5. TUBERCULOSIS 1.7
6. MEASLES 1.1
7. SCARLET FEVER DIPHTHERIA TYPHOID AND PARATYPHOID 1.35
SUMMER
5 MONTHS APR.'18-AUG.'18
0 10 20 30 40 50 60 70 80 90
1. PNEUMONIA 49.3
2. DISEASES OTHER THAN HERE MENTIONED 22.0
3. MENINGITIS (ALL KINDS) 7.3
4. CAUSES OTHER THAN DISEASE 16.4
5. TUBERCULOSIS 3.2
6. MEASLES .56
7. SCARLET FEVER DIPHTHERIA TYPHOID AND PARATYPHOID 1.49
AUTUMN
4 MONTHS SEPT.'18-DEC.'18
0 10 20 30 40 50 60 70 80 90
1. PNEUMONIA AND INFLUENZA 93.7
2. DISEASES OTHER THAN HERE MENTIONED 2.6
3. MENINGITIS (ALL KINDS) 1.0
4. CAUSES OTHER THAN DISEASE 1.6
5. TUBERCULOSIS .35
6. MEASLES .059
7. SCARLET FEVER DIPHTHERIA TYPHOID AND PARATYPHOID .158

Chart IV.

be charged to some extent to the hastening of the disease by an attack of influenza. For the group of diseases which have not been treated separately, and in which we have placed influenza on the chart, there was a slight reduction during the summer and then the number increased threefold. There was not much variation in the deaths from "causes other than disease," the greatest figure occurring in the summer. The combined effect of these various causes is clear in the diagram at the bottom of the chart where there is shown the annual death rate per 1,000 from all causes for each season. The drop from 9.1 during the winter to 5.7 for the summer was offset by the enormous rise during the autumn to 48.5.

Comparison of the fatality of the disease within the seasons has been illustrated in Chart IV, the graph in this case showing the percentage of deaths from each cause. Outside the variation in the pneumonia column which is the most striking feature of this chart, we may call attention to the relative increase in the deaths from "diseases other than here mentioned" and the "causes other than disease" during the summer season. The deaths from these two causes were greater in proportion to the total deaths during the summer than during the previous winter or the autumn. The three diseases, scarlet fever, diphtheria and typhoid (and paratyphoid) make up approximately 1 per cent of all deaths.

A rate is significant only in contrast to some other rate. The army death rates may be more clearly understood if we show what has happened in civilian life. Our most accurate civilian rates are to be found by going back to the year 1910, for this was the census year, and the mortality and population statistics are more reliable than in the years following when populations have to be estimated. In Table IV we have placed side by side for the different diseases

TABLE 4

COMPARISON OF DEATH RATES OF ARMY AND CIVILIAN MALES, AGE 20 TO 29 YEARS
ANNUAL RATE PER 100,000

DISEASE	ARMY	CIVILIAN MALES
Pneumonia	*431.	45.5
Meningitis	70.6	4.6
Scarlet fever	4.7	1.9
Diphtheria	2.2	2.0
Typhoid fever	2.8	47.2
Measles	5.6	.94
Tuberculosis	24.4	206.
All causes	*760.	642.

NOTE: Army Period is average of time from October, 1917, to December, 1918, inclusive. Civilian Period is year 1910.

*The "Autumn Season" has been omitted in the Army Rates for Pneumonia and All Causes because of the influenza epidemic for which there is no counterpart in civilian life during 1910.

both the army rate and the rate for civilian males in the Registration Area. This army rate is made up from a weighted average of the winter, summer and autumn seasons. In the case of pneumonia and death from all causes, the autumn rate has been omitted. This was the influenza season and the deaths under these two headings are abnormal, and there is nothing comparable in 1910 in the civilian rates. The civilian rate is made up of males in the age group twenty

to twenty-nine years, as this is the fairest comparison with the army. These rates are expressed in terms of annual rate per 100,000.

Added significance is given to this table by expressing the rates from the two sources in terms of a ratio as is done below:

Dividing one rate by the other we see that—

Pneumonia is 9 times greater in the army.
Meningitis is 15 times greater in the army.
Scarlet fever is 2.5 times greater in the army.
Diphtheria is 1.1 times greater in the army.
Measles is 6 times greater in the army.
Deaths from all causes are 1.2 times greater in the army.
Tuberculosis is 8 times greater in civilian life.
Typhoid fever is 17 times greater in civilian lfe.

There is a wealth of significance in the relationship expressed above.

Tuberculosis and typhoid fever are the two diseases in which the army far excels civilian life. Tuberculosis is less in the army because the tuberculous individual has for the most part been prevented by the medical examination from entering the army. Typhoid fever is less in the army because every man is given a protective typhoid inoculation and because water supplies are under close supervision.

Diphtheria and scarlet fever prevail in the army in about the same degree as in civilian life.

Measles is much greater in the army because susceptible people have been drawn in from rural territory and brought in contact with the disease in camp. In other words, the exposure of susceptibles has been increased upon entering the service.

Pneumonia is greater in the army probably for two reasons: One is that the susceptible person has been placed in more frequent proximity with the individual who habitually harbors these organisms in his throat. The second is that the hardening process of camp life is a severe strain upon many constitutions, especially in the first few weeks. Cold, exposure, and fatigue are sufficient to lower the person's natural resistance to a point where either his own pneumococci become harmful or where the pneumonia-causing germs from other individuals infect him.

Meningitis has not appeared in the army as an epidemic disease. It is true that some camps have had many more cases than others, but the cases are never localized in any one organization. Rather do these spring up sporadically at widely separated points. The fact that meningitis has been fifteen times as prevalent in the army as among young civilian males is explained by the enormous multiplication of human contacts in camp. It is probable that few people are susceptible to meningitis. We know that meningococcic carriers exist. In camp the carriers rub elbows with some of this susceptible population. Meningitis occurs for the most part in the cold or changeable seasons. The greater exposure which the soldier undergoes in camp makes colds more frequent. Secretions from the sinuses pour down in greater quantity than normally, and become smeared over the hands of the soldier. As Lieut.-Col. Hans Zinsser has

so clearly stated, the common colds increase the secretion from the upper sinuses bringing down into the nasopharynx bacterial matter, which would not happen if the colds were absent. In this way it is probable that many people whose secretions are normally free from the meningococcus become dangerous carriers when the secretions from these deeper pockets are loosened. The problem facing army sanitarians is revealed in Table 4. It is pneumonia and meningitis against which the greatest efforts must be directed.

B. CAUSES OF SICKNESS

The most frequent causes of sickness serious enough to make the soldier take to his bed are those minor infections such as colds, grippe, mumps, bronchitis, etc. During the summer season there were almost 530,000 admissions for this group of diseases. Expressed in terms of annual rate per 1,000 this would be about 882. The rate for venereal disease was 156, which is about one-fifth of that of the above group of diseases. Measles stands next in frequency, and this is followed by pneumonia, malaria, and scarlet fever. Last come meningitis, dysentery, and typhoid. Admissions for disease represent 94 per cent of the total admissions. The absolute number of admissions and the rate per 1000 will be found in Table 5.

TABLE 5

CAUSES OF ADMISSION TO HOSPITAL AND QUARTERS

All Troops in United States

Five Summer Months, Week Ending April 5, 1918, to August 30, 1918
Average Strength—1,420,000

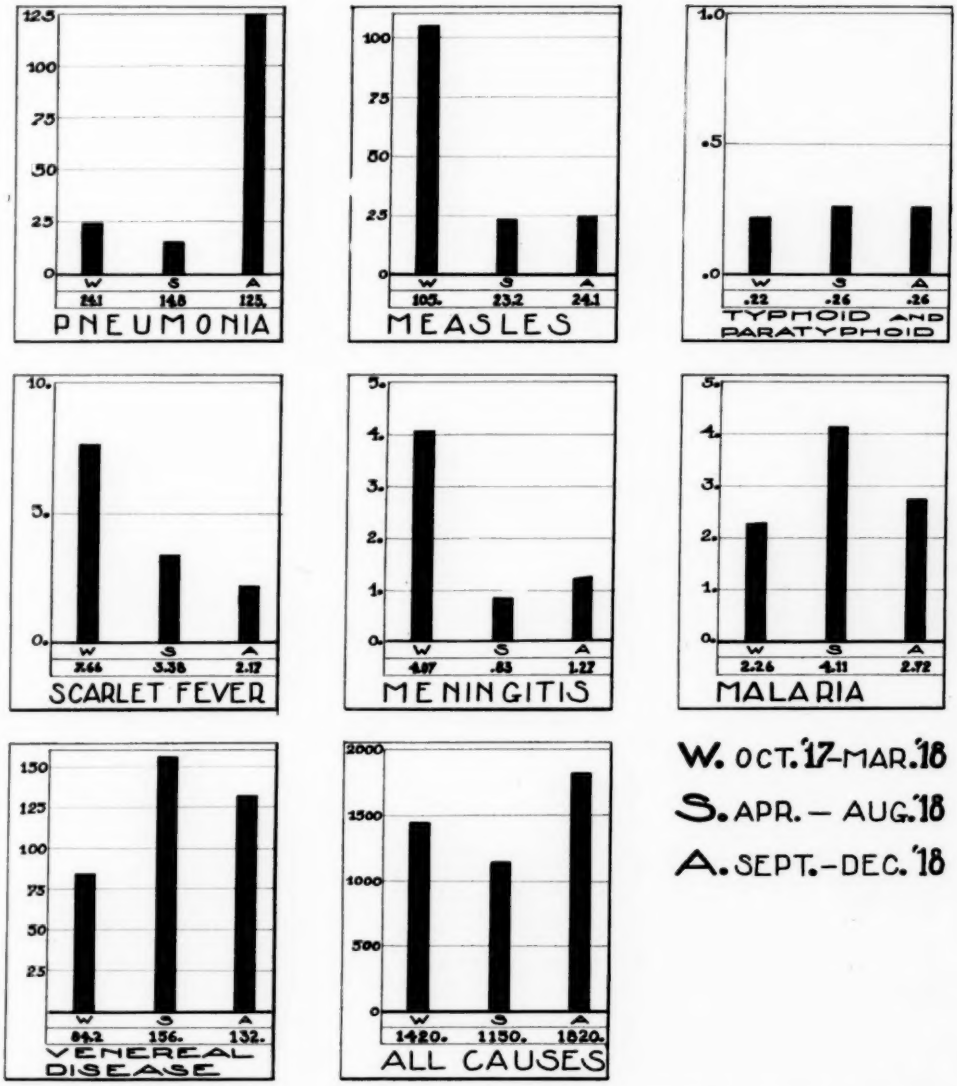
DISEASE	NUMBER OF ADMISSIONS	ANNUAL RATE PER 1000
Disease other than here mentioned*	529,887	882.
Venereal	93,959	156.
Measles	13,949	23.2
Pneumonia	8,912	14.8
Malaria	2,467	4.11
Scarlet fever	2,029	3.38
Meningitis	497	.83
Dysentery	496	.83
Typhoid and paratyphoid fever	156	.26
Total disease	652,352	1082.
Causes other than disease**	39,722	66.0
All causes	692,074	1148.

*Includes minor affections such as colds, la grippe, mumps, whooping cough, bronchitis, influenza, etc., and the rarer diseases like smallpox, anthrax, tetanus, etc.
**Includes fractures, sunstroke, gunshot wounds, etc.

During the autumn season we find for the miscellaneous group of diseases only 381,000 admissions, which gives an annual rate of 778. This is less than the rate for the summer. However, the admissions for influenza, which were included in the miscellaneous group during the summer, amounted during the autumn to 338,000. If this were added to the miscellaneous group in the autumn, the admission rate would amount to about 1,400. The fact that the admission rate for the miscellaneous group, exclusive of influenza, was less in the

SICKNESS AMONG ALL TROOPS IN U.S. BY SEASONS. 1917-1918

ANNUAL RATE PER 1000



W. OCT. '17-MAR. '18

S. APR. – AUG. '18

A. SEPT.-DEC. '18

CHART SHOWS CAUSE OF ADMISSION TO HOSPITAL AND QUARTERS FOR ALL TROOPS IN THE UNITED STATES.

Chart V.

autumn indicates that many of the minor illnesses were avoided, the persons who might have had these having influenza instead. The greatest difference between autumn and summer so far as admissions are concerned, is in pneumonia. The summer rate had been 14.8. The rate in the autumn was 125. Next to pneumonia comes measles with a rate of 24.1. After this come malaria, scarlet fever and meningitis, the table concluding with dysentery and typhoid. Disease during the autumn was responsible for 95 per cent of the total admissions. These data are given in Table 6.

That we may better appreciate the significance of these admission rates we have shown in Chart V the rates for each disease by seasons, comparing one season with another. This picture is not greatly different from the chart of deaths. The pneumonia cases dropped off in the summer and increased enormously in the autumn. The greatest measles rate occurred in the winter season, dropped to about one-fifth of this figure in the summer and remained there during the autumn. Scarlet fever decreased progressively, the autumn rate being about one-third that of the previous winter. Meningitis dropped appreciably in the summer, the winter and autumn rates being about the same. Venereal dis-

TABLE 6

CAUSES OF ADMISSION TO HOSPITAL AND QUARTERS

All Troops in United States

Four Autumn Months, Week Ending September 6, 1918, to December 27, 1918
Average Strength—1,493,000

DISEASE	NUMBER OF ADMISSIONS	ANNUAL RATE PER 1000
Disease other than here mentioned*	380,928	778.
Influenza	338,342	690.
Venereal	64,672	132.
Pneumonia	61,191	125.
Measles	11,789	24.1
Malaria	1,331	2.72
Scarlet fever	1,060	2.17
Meningitis	619	1.27
Dysentery	237	.48
Typhoid and paratyphoid fever	131	.27
Total disease	850,301	1740.
Causes other than disease**	40,442	82.6
All causes	890,743	1823.

*Includes minor affections such as colds, la grippe, mumps, whooping cough, bronchitis, etc., and the rarer diseases like smallpox, anthrax, tetanus, etc. Influenza is no longer carried as a "minor affection."

**Includes fractures, sunstroke, gunshot wounds, etc.

ease was greater in the summer and dropped off slightly in the autumn. For the miscellaneous group of diseases the rate is lowest in the summer. This takes a great rise in the autumn due to influenza. At the bottom of the chart is a graph showing the rates for all causes of admission, over 90 per cent being due in each case to disease. It is surprising not to find the autumn rate much larger than it is, in view of the influenza epidemic. It is not larger because the miscellaneous group of diseases, which makes up the bulk of the admissions,

is so great normally that additional admissions for specific causes affect the total rate but slightly.

In order to understand the variation that occurs in the different seasons in the factors which send men to the hospital, we have expressed in Chart VI, the percentage of distribution of admissions by seasons. The most noticeable features in this chart are the falling off of measles, during the summer months, the increase of venereal disease during the summer months, the position of measles in the autumn, and the increase of pneumonia in the autumn. Scarlet fever, meningitis, malaria and typhoid, together caused less than 1 per cent of all hospital admissions each season.

C. CASE MORTALITY

Some idea of the seriousness of disease in the army and the variations in fatality from season to season may be gained by placing the cases and deaths from each cause side by side and computing therefrom the case mortality. This we have done in Table 7. We have grouped the records of the three seasons

TABLE 7

CASE MORTALITY RATES FOR CERTAIN DISEASES AT DIFFERENT PERIODS OF 1917 AND 1918

(All Troops in United States)

DISEASE	SEASON		CASES	DEATHS	% FATALITY
	1917		8,479	952	11.2
Pneumonia	Winter	17-18	13,393	3,100	23.1
	Summer	'18	8,912	1,679	18.8
	Autumn	'18	61,191	21,053	34.4
Scarlet Fever	1917		1,966	48	2.4
	Winter	17-18	4,253	40	0.9
	Summer	'18	2,029	26	1.3
	Autumn	'18	1,060	10	1.1
Measles	1917		44,158	20	0.05
(uncomplicated)	Winter	17-18	58,035	52	0.09
	Summer	'18	13,949	19	0.14
	Autumn	'18	11,789	14	0.12
Diphtheria	1917		1,163	12	1.03
	1917		1,027	325	31.7
Meningitis	Winter	17-18	2,264	604	26.6
(all forms)	Summer	'18	497	248	50.0
	Autumn	'18	619	244	39.5
Typhoid and	1917		238	18	7.6.
paratyphoid	Winter	17-18	124	7	5.6
	Summer	'18	156	20	12.8
	Autumn	'18	131	21	16.0
Influenza	1917		32,248	7	.02
	Autumn	'18	338,343	1,133	.34

NOTE: With the exception of the year 1917, these figures should be accepted with caution. They represent the number of cases and number of deaths within a definite period. Cases and deaths do not necessarily represent the same group of people. Some deaths may result from cases reported in a previous season. Other deaths may occur in the season following the case record and thus be omitted. For the true facts we must await the analysis of the Sick and Wounded cards.

and the figures for the year 1917 which are taken from the Surgeon General's Annual Report. A number of interesting facts are brought out in this table. In the first place we see a variation from season to season in the case mortality. During the entire year 1917 the case mortality rate for pneumonia was 11.2 per cent. During the winter months this rate increased to 23.1 per cent, more than

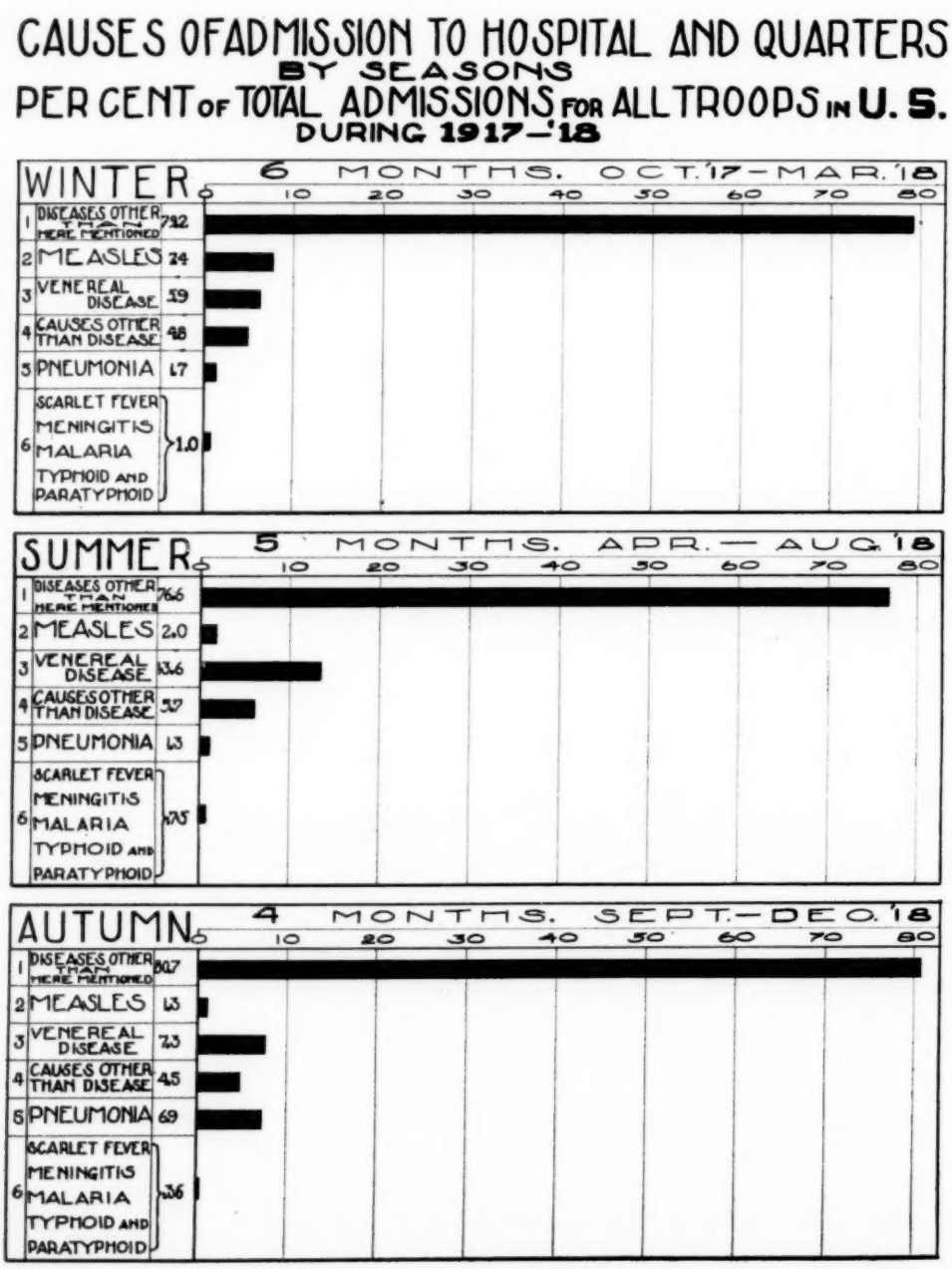

CAUSES OF ADMISSION TO HOSPITAL AND QUARTERS
BY SEASONS
PER CENT OF TOTAL ADMISSIONS FOR ALL TROOPS IN U. S.
DURING 1917–'18
WINTER
6 MONTHS. OCT. '17 – MAR. '18
0 10 20 30 40 50 60 70 80
1 DISEASES OTHER THAN HERE MENTIONED 73.2
2 MEASLES 2.4
3 VENEREAL DISEASE 3.9
4 CAUSES OTHER THAN DISEASE 4.8
5 PNEUMONIA 1.7
6 SCARLET FEVER, MENINGITIS, MALARIA, TYPHOID AND PARATYPHOID 1.0
SUMMER
5 MONTHS. APR. — AUG. '18
0 10 20 30 40 50 60 70 80
1 DISEASES OTHER THAN HERE MENTIONED 76.6
2 MEASLES 2.0
3 VENEREAL DISEASE 3.6
4 CAUSES OTHER THAN DISEASE 3.7
5 PNEUMONIA 1.3
6 SCARLET FEVER, MENINGITIS, MALARIA, TYPHOID AND PARATYPHOID .75
AUTUMN
4 MONTHS. SEPT. – DEC. '18
0 10 20 30 40 50 60 70 80
1 DISEASES OTHER THAN HERE MENTIONED 80.7
2 MEASLES 1.3
3 VENEREAL DISEASE 2.3
4 CAUSES OTHER THAN DISEASE 4.5
5 PNEUMONIA 6.9
6 SCARLET FEVER, MENINGITIS, MALARIA, TYPHOID AND PARATYPHOID .36

Chart VI.

double. There is an overlapping in these two seasons, three of the winter months being included in the record for the year 1917. The winter of 1917-18 was characterized by epidemics of measles and bronchopneumonia. It is these two causes, mainly the latter, which are responsible for the increased case mortality in the second period. As pneumonia decreased during the suummer, the severity of the disease likewise lessened and the case mortality dropped to 18.8 per cent. During the influenza period in the autumn of 1918, bronchopneumonia increased to a tremendous degree and not only did the absolute number of cases increase, but the case mortality rose to 34.4 per cent.

Meningitis shows a wide variation from season to season. The case mortality rate for 1917 was 31.7 per cent. During the winter of 1917-18 this dropped to 26.6 per cent. During the summer season it rose to 50 per cent. The months of April and May are included in the summer group and it is the experience in civil life that meningitis is most prevalent in the spring. In the autumn the case mortality dropped to 39½ per cent, which is higher than the rates for the first two periods.

The case mortality for typhoid and paratyphoid was 7.6 per cent during the year 1917. This dropped to 5.6 during the winter of 1917-18, then rose to 12.8 per cent in the summer and to 16 per cent in the autumn. Scarlet fever shows the highest case mortality during the year 1917. This figure dropped to nearly ⅓ this rate during the winter months and remained only slightly above this level for the remainder of the year.

The measles deaths are so few in number and so small as compared to the cases that the case mortality rates are without much significance. The only point of interest is that the rates were higher during the summer and autumn than they were during the previous winter or the previous year although the measles epidemic period is included in these latter periods. Considering the changes in the case mortality rates from season to season, we find no similarily in the different diseases. No disease had its highest rate during the winter of 1917-18. The summer of 1918 saw the highest rate in meningitis and measles. The autumn of 1918 marked high rates for pneumonia and typhoid.

D. WEEKLY INCIDENCE OF DEATHS AND DISEASE AMONG TROOPS IN THIS COUNTRY COMPARED WITH THE EXPEDITIONARY FORCE

The figures given above represent summaries for entire seasons. It is interesting to follow the disease rates from week to week. It is possible in this connection to compare rates for disease and death among the troops in this country with the rates for the American Expeditionary Force. The weekly rates expressed as an annual rate per thousand are given in Table 8.

In the first column appear deaths from all causes and these figures have been illustrated in Chart VII. The troops in this country show a higher rate than the the Expeditionary Force during November and December and up to the first week in January. From this time rates for the Expeditionary Force forge ahead and remain greater up to the second week in October. It is at this time that the influenza epidemic was at its height in this country and for two weeks the home troops unexposed to rifle fire died off at a greater rate than the men abroad who were exposed to hardship and the bullets of the enemy. Rates

for the Expeditionary Force following the third week in October are higher than that for the troops in this country and remain so for the remainder of the year.

"Death from Disease Only" is illustrated in Chart VIII. It will be noted that the troops at home had higher rates than those abroad up to the second week

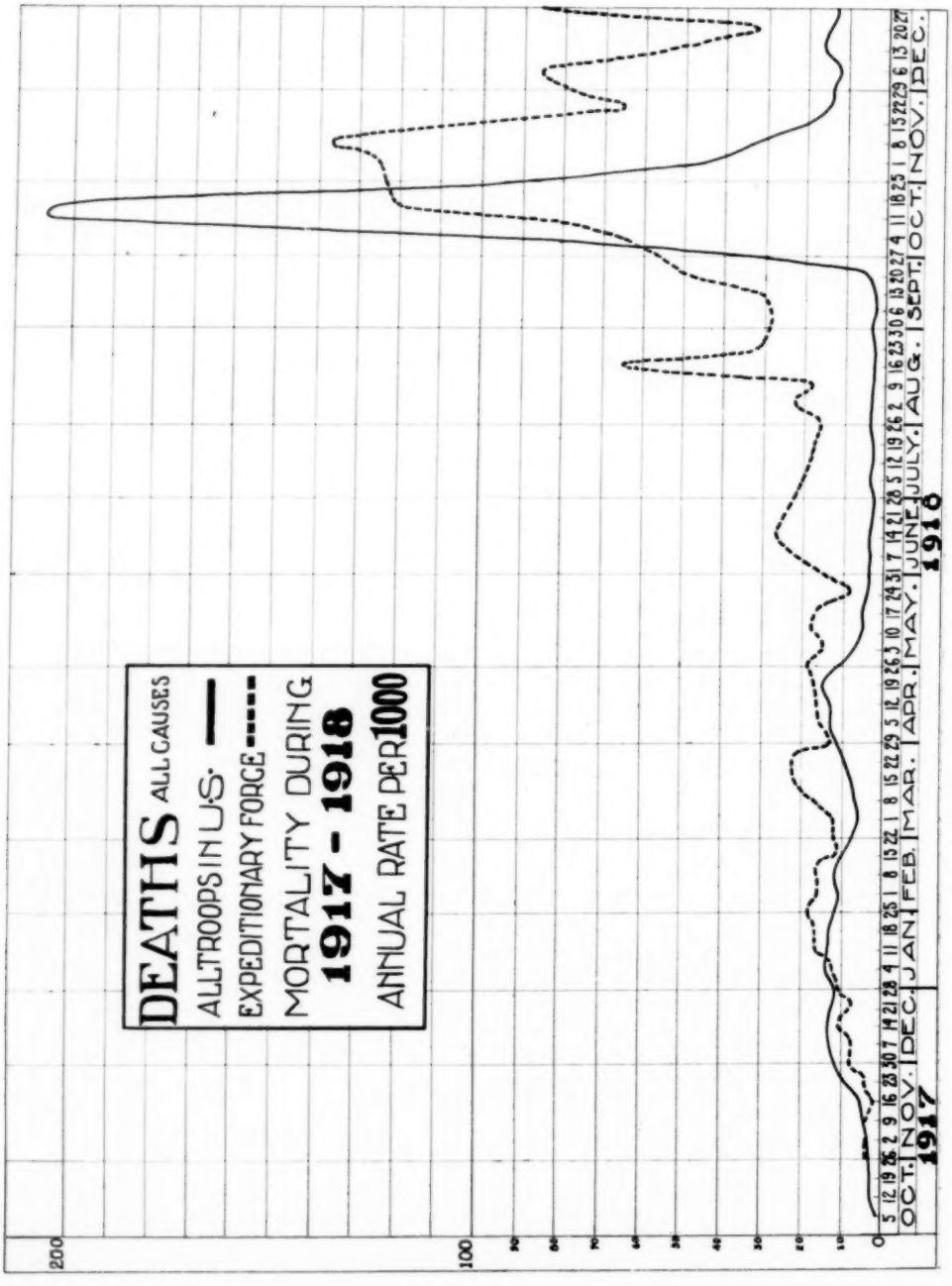


Chart VII.

in January. The leadership is then reversed for several weeks, but by the last week in March, rates for the men in this country again exceed those for the men abroad and this relationship continues with but here and there an interruption for the rest of the year. This is a striking fact and one that demands serious thought from those who are charged with the duty of recruiting armies.

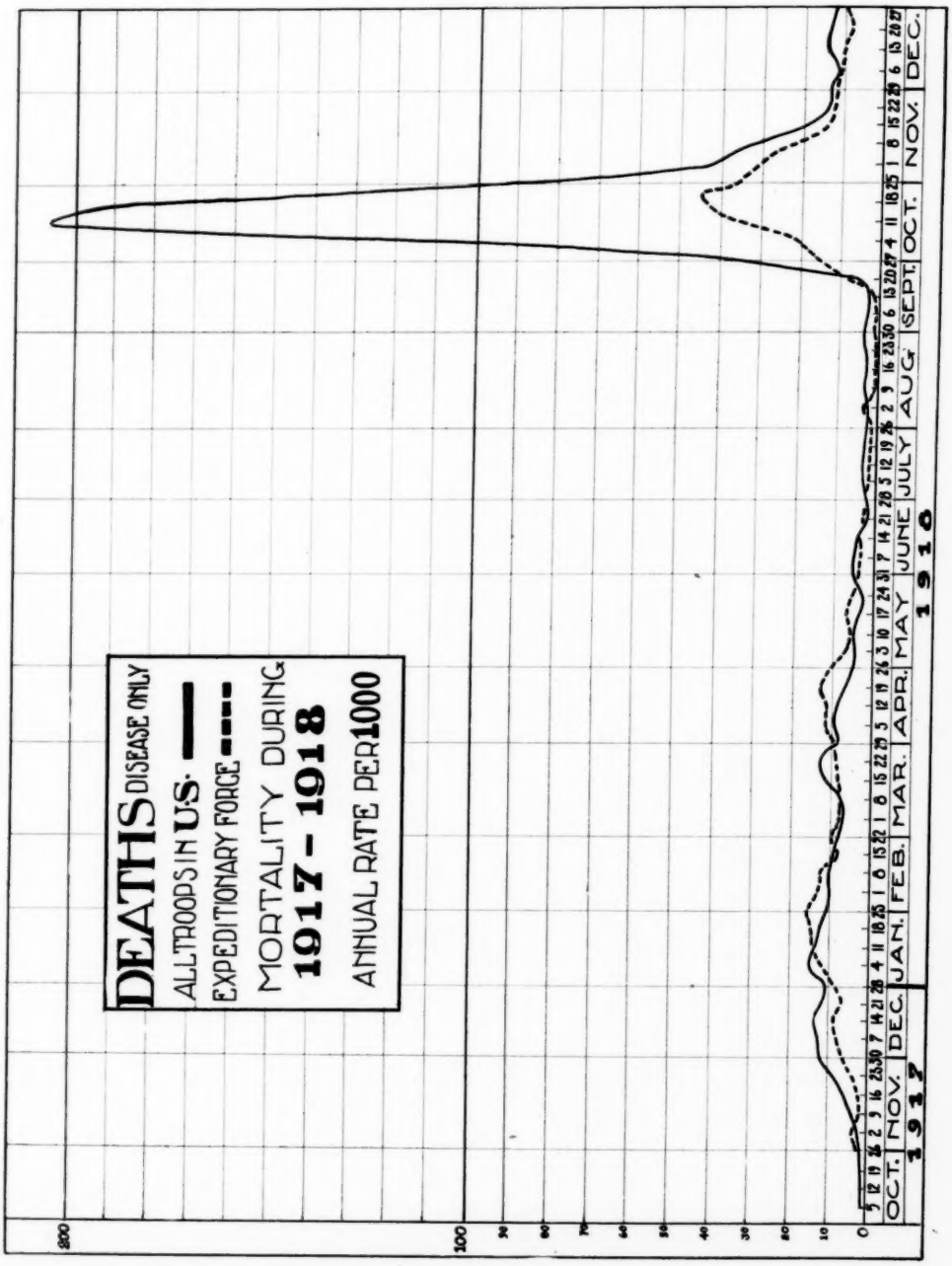


Chart VIII.

TABLE 8

DEATH AND DISEASE IN U. S. ARMY DURING THE GREAT WAR

Weekly Incidence for Troops at Home and Abroad from October, 1917, to December, 1918

Annual Rate per 1,000

WEEK ENDING	DEATHS				ADMISSIONS FOR DISEASE																	
	ALL CAUSES		DISEASE ONLY		PNEUMONIA		DYSENTERY		MALARIA		VENEREAL		TYPHOID&PARA.		MEASLES		MENINGITIS		SCAR. FEV.	
	U.S.	A.E.F.	U.S.	A.E.F.	U.S.	A.E.F.	U.S.	A.E.F.	U.S.	A.E.F.	U.S.	A.E.F.	U.S.	A.E.F.	U.S.	A.E.F.	U.S.	A.E.F.	U.S.	A.E.F.
1917																				
Oct. 5	1.6	..	1.1	..	4.0	..	2.8	..	13.1	..	182.0	..	0.2	..	19.9	..	.21	..	1.5	..
12	2.0	..	1.7	..	4.8	..	3.2	..	8.3	..	118.0	..	0.4	..	24.0	..	.8	..	0.8	..
19	2.4	..	1.9	..	10.4	..	.7	..	5.2	..	114.0	..	0.6	..	26.0	..	.6	..	0.9	..
26	2.8	4.03	2.1	2.7	13.7	5.4	1.0	..	7.5	..	93.0	54.5	0.7	0.7	54.0	12.1	1.8	0.7	1.1	2.7
Nov. 2	2.9	3.7	2.1	3.7	11.8	23.7	0.5	4.3	4.8	1.2	107.0	202.	0.4	1.9	113.	8.1	1.8	0.0	2.0	1.8
9	4.8	4.1	4.1	2.0	20.4	16.7	0.8	2.1	3.6	1.0	117.0	182.	0.5	0.0	242.	27.7	2.8	0.0	1.6	1.0
16	5.9	2.7	5.1	2.2	23.1	7.2	0.3	0.0	3.0	0.9	88.0	81.1	0.9	0.0	317.	31.5	2.3	0.0	2.4	1.3
23	9.3	4.4	8.9	3.1	30.8	21.2	0.8	0.8	3.0	5.8	101.0	82.5	0.2	1.3	292.	89.2	2.9	1.7	2.7	8.4
30	12.8	8.1	12.3	6.0	22.6	14.2	0.3	0.4	2.6	0.0	101.0	75.8	0.4	0.0	176.	60.3	2.8	3.8	5.3	3.0
Dec. 7	13.3	8.3	12.6	7.0	30.9	10.7	0.1	0.0	0.9	1.5	81.0	71.0	1.1	0.3	223.0	69.0	3.8	7.9	5.1	2.7
14	13.6	11.4	13.4	8.1	31.9	25.1	0.6	1.8	1.3	1.1	63.0	122.	0.05	0.4	189.0	49.4	4.3	3.6	6.2	14.0
21	12.3	7.7	11.7	6.3	29.9	43.7	0.2	0.7	1.0	0.3	67.0	94.9	0.0	0.0	156.0	34.9	4.5	5.5	6.7	18.4
28	11.5	11.0	10.6	8.0	33.9	21.4	0.1	1.6	0.6	0.0	64.0	74.8	0.1	0.3	122.0	43.9	4.8	5.0	6.0	6.7
1918																				
Jan. 4	14.5	13.3	14.7	11.6	28.7	54.3	0.5	1.1	.7	0.6	68.0	50.0	0.04	0.0	108.0	55.8	4.4	5.2	7.4	9.0
11	13.7	16.4	13.2	13.6	38.7	52.9	0.2	1.7	1.3	0.8	81.0	57.5	0.1	0.6	109.0	77.5	4.4	2.6	8.4	27.7
18	13.5	16.4	12.9	14.2	33.0	82.2	0.4	1.1	.7	0.2	71.0	74.3	0.04	0.4	108.0	63.4	4.0	5.7	11.2	18.4
25	11.4	17.8	10.7	15.9	36.6	59.6	0.4	2.9	.9	0.0	69.0	52.6	0.1	0.7	92.0	66.3	5.3	6.7	9.3	27.5
Feb. 1	10.9	15.4	10.0	13.1	36.1	30.0	0.4	1.0	1.4	1.0	84.0	57.3	0.0	0.0	77.0	48.4	3.6	6.3	12.7	12.1
8	11.6	16.0	10.9	12.2	35.8	33.5	0.2	0.7	1.0	1.2	70.0	57.9	0.0	0.2	57.0	25.8	5.3	1.6	14.6	13.4
15	10.3	11.6	9.7	7.8	30.6	18.5	0.2	0.2	0.2	0.2	69.0	51.7	0.0	0.2	48.0	26.4	3.2	4.2	12.1	11.1
22	8.8	12.3	8.4	9.7	21.2	21.3	0.3	0.4	0.8	0.4	70.7	55.3	0.1	0.0	42.9	26.9	2.8	6.0	13.8	10.5
Mar. 1	6.7	12.4	6.6	7.0	19.7	26.0	0.2	0.2	0.5	0.0	97.1	44.2	0.2	0.4	34.7	13.7	1.5	3.0	12.0	13.4
8	7.2	19.0	6.6	6.6	18.2	36.5	0.3	1.0	0.9	0.4	100.	48.5	0.0	0.0	36.7	16.2	1.6	6.6	10.7	21.2
15	7.6	22.4	7.5	11.1	21.0	36.2	0.2	0.7	1.3	0.0	90.6	46.0	0.0	0.2	33.7	16.5	1.6	2.5	13.2	30.9
22	9.7	23.5	8.9	12.6	23.5	46.4	0.1	0.5	1.9	0.2	78.5	59.0	0.1	0.2	34.4	8.9	1.6	3.5	11.1	22.6
29	10.7	13.5	9.8	8.0	31.7	40.4	0.3	0.5	1.3	0.0	74.3	48.7	0.0	0.0	31.5	9.2	2.1	1.7	11.4	15.2
Apr. 5	12.2	16.5	11.6	9.8	26.6	35.8	0.3	1.0	2.7	0.0	129.	49.2	0.04	0.4	29.5	10.3	1.5	3.3	11.1	13.7
12	12.2	17.3	11.3	8.8	34.6	27.8	0.4	0.6	1.7	0.4	103.	47.9	0.04	0.3	28.0	4.4	1.4	2.4	12.1	15.9
19	14.3	18.1	12.9	6.4	33.8	21.3	0.1	0.0	1.9	0.7	86.9	52.8	0.0	0.1	25.7	5.2	1.8	3.4	10.8	6.7
26	10.9	19.2	10.4	4.5	26.6	28.4	0.2	0.1	2.2	1.3	78.9	40.5	0.2	0.0	21.9	6.2	1.0	2.3	8.3	9.7

TABLE 8—Continued

DEATH AND DISEASE IN U. S. ARMY DURING THE GREAT WAR

Weekly Incidence for Troops at Home and Abroad from October, 1917, to December, 1918

Annual Rate per 1,000

WEEK ENDING	DEATHS: ALL CAUSES U.S.	ALL CAUSES A.E.F.	DISEASE ONLY U.S.	DISEASE ONLY A.E.F.	PNEUMONIA U.S.	PNEUMONIA A.E.F.	ADMISSIONS FOR DISEASE: DYSENTERY U.S.	DYSENTERY A.E.F.	MALARIA U.S.	MALARIA A.E.F.	VENEREAL U.S.	VENEREAL A.E.F.	TYPHOID&PARA. U.S.	TYPHOID&PARA. A.E.F.	MEASLES U.S.	MEASLES A.E.F.	MENINGITIS U.S.	MENINGITIS A.E.F.	SCAR.FEVER U.S.	SCAR.FEVER A.E.F.
May 3	7.0	15.1	6.3	4.2	16.6	23.5	0.5	0.7	2.6	1.2	146.	35.1	0.04	0.2	22.9	8.5	1.4	2.4	5.8	8.3
10	6.6	18.0	5.3	4.6	20.0	21.7	0.7	0.1	2.4	1.1	183.	32.3	0.3	0.2	21.1	9.3	1.4	2.1	8.1	6.4
17	7.1	17.3	6.3	3.3	21.8	12.8	0.6	0.3	2.9	1.0	157.	37.8	0.0	0.0	21.0	12.3	1.3	1.4	6.4	5.7
24	6.6	8.5	5.7	2.2	14.4	20.8	1.0	1.7	2.7	0.8	129.0	75.4	0.1	0.9	18.3	9.7	1.0	1.1	3.4	3.0
31	4.9	16.1	4.0	5.4	7.9	15.0	1.4	0.43	4.2	1.0	176.	38.3	0.08	0.08	19.2	8.6	0.9	2.5	3.1	7.2
June 7	4.1	22.1	3.2	4.6	8.1	10.8	0.7	0.2	4.0	0.9	175.	38.4	0.14	0.08	15.5	8.5	1.2	1.7	2.0	2.2
14	4.3	26.0	3.3	3.7	8.6	10.0	0.9	0.0	4.7	1.2	161.	47.8	0.04	0.2	21.0	6.0	.7	2.1	1.3	3.9
21	3.4	24.1	2.9	2.3	9.6	8.6	1.6	0.3	6.9	1.3	134.	33.0	0.5	0.3	20.8	4.5	.7	1.1	1.4	2.9
28	3.0	22.2	2.1	2.1	8.5	9.2	1.5	0.7	6.6	1.2	133.	29.8	0.4	0.0	23.6	6.1	.8	1.6	1.3	4.9
July 5	4.3	20.2	3.2	1.9	7.5	9.02	0.6	0.22	6.1	0.9	189.	24.5	0.3	0.3	25.8	10.7	.8	0.9	1.5	2.2
12	3.7	19.9	3.0	1.9	6.6	8.1	0.7	0.4	7.3	0.8	145.	23.5	0.4	0.2	30.2	4.1	.7	1.7	1.8	2.1
19	3.6	18.1	2.9	1.7	10.1	9.3	1.0	0.6	5.6	0.7	167.	27.2	0.4	0.2	26.5	2.1	.6	0.9	0.9	0.9
26	3.7	16.9	2.1	1.6	9.3	9.4	1.0	2.3	5.7	1.2	182.	29.8	0.7	0.9	26.4	7.8	.5	0.9	0.5	1.9
Aug. 2	3.6	22.7	2.5	2.8	9.5	5.6	0.9	2.7	4.5	0.9	162.	21.9	0.4	1.8	21.9	5.0	.4	0.8	0.3	1.0
9	4.1	18.5	3.2	0.3	8.5	6.3	0.9	2.9	5.2	0.9	218.	19.8	0.5	0.3	25.0	5.0	.3	0.7	0.4	1.3
16	3.8	66.7	2.7	0.4	10.8	6.5	0.8	2.7	6.0	0.7	232.	20.1	0.4	0.8	24.0	6.5	.2	0.8	0.5	0.9
23	3.5	33.1	3.0	0.2	11.6	8.5	1.3	3.4	5.2	1.2	200.	19.5	0.4	1.4	26.6	11.6	.2	1.1	0.3	1.3
30	4.1	44.9	3.4	0.8	14.7	11.5	1.0	7.9	5.6	0.8	171.	19.4	0.2	0.8	26.1	5.9	.5	0.6	0.4	0.0
Sept. 6	3.2	29.9	2.7	0.3	11.3	10.4	0.4	5.2	5.1	0.4	313.	21.0	0.4	0.4	20.7	5.2	.5	0.8	0.3	0.5
13	3.3	30.9	2.4	1.1	10.2	16.5	0.8	4.8	6.4	0.8	321.	22.8	0.8	0.9	21.5	9.2	.4	0.5	0.3	0.3
20	5.4	49.6	4.5	7.9	24.1	27.3	0.6	4.1	5.4	0.6	177.	26.6	0.5	0.7	22.6	9.8	.5	0.5	1.6	0.9
27	33.2	55.5	32.5	14.6	138.	37.5	0.8	5.8	5.2	0.6	106.	23.8	0.4	0.6	26.2	8.2	.5	1.7	0.6	1.2
Oct. 4	82.7	80.5	81.8	20.1	282.	71.0	0.3	4.4	2.9	0.5	124.	18.8	0.1	0.8	22.7	6.2	.9	2.1	0.6	0.8
11	207.0	80.1	206.	41.6	592.	86.8	0.4	4.0	1.7	0.6	70.3	17.0	0.2	0.2	21.1	6.1	1.5	3.5	0.9	0.6
18	191.0	122.	190.	44.7	371.	107.	0.4	3.1	1.5	0.3	81.2	19.6	0.1	0.3	28.5	6.9	3.7	5.9	0.6	7.2
25	93.2	122.	92.7	35.0	202.	67.2	0.3	5.3	1.9	0.5	83.1	16.0	0.2	0.2	24.2	8.5	3.8	3.3	1.5	0.9
Nov. 1	44.0	126.	43.2	30.8	98.9	56.7	0.7	3.8	2.1	0.4	85.9	21.6	0.1	0.5	31.4	10.3	2.0	3.3	0.9	1.2
8	34.0	139.	33.1	21.5	81.1	40.8	0.2	6.5	1.6	0.3	173.	21.0	0.1	0.6	29.4	6.2	0.9	1.6	3 3	0.8
15	20.3	111.	19.8	12.9	45.0	33.1	0.2	4.1	2.2	0.4	191.	20.5	0.2	0.3	27.7	9.1	1.0	1.6	8.4	0.7
22	14.7	66.5	13.8	12.7	36.4	38.0	0.5	4.0	1.5	0.4	156.	18.5	0.3	0.8	26.8	7.9	0.6	2.0	4.7	1.1
29	14.1	79.6	13.3	12.6	38.7	34.21	0.4	4.0	0.8	0.4	53.5	20.4	0.1	0.3	23.4	14.5	0.6	1.6	3.	0.7
Dec. 6	12.7	85.4	11.7	11.5	39.7	31.6	0.7	2.4	1.1	0.2	31.0	21.5	0.3	0.8	23.1	5.1	1.2	2.2	2.6	0.8
13	15.5	55.1	14.9	10.4	41.4	26.6	0.2	4.8	1.2	0.2	54.7	30.1	0.03	1.3	22.2	5.8	1.4	1.9	2.5	1.4
20	14.7	32.2	13.7	7.5	37.0	30.7	0.5	1.5	2.4	0.2	64.2	26.7	0.0	2.1	21.1	6.7	1.1	2.9	3.6	1.1
27	12.9	89.8	12.2	9.2	26.4	24.4	0.1	2.4	1.6	0.6	52.4	23.1	0.0	1.0	14.3	3.6	1.1	1.9	2.5	1.0

There is more disease among the home troops because their vital resistance is reduced by the transition from civilian to army life. The rates for the Expeditionary Force are low because the new recruit and the weakling do not go across to France. The weakling is weeded out before he leaves and the new recruit becomes a seasoned soldier by the time he sails. The hardships of war

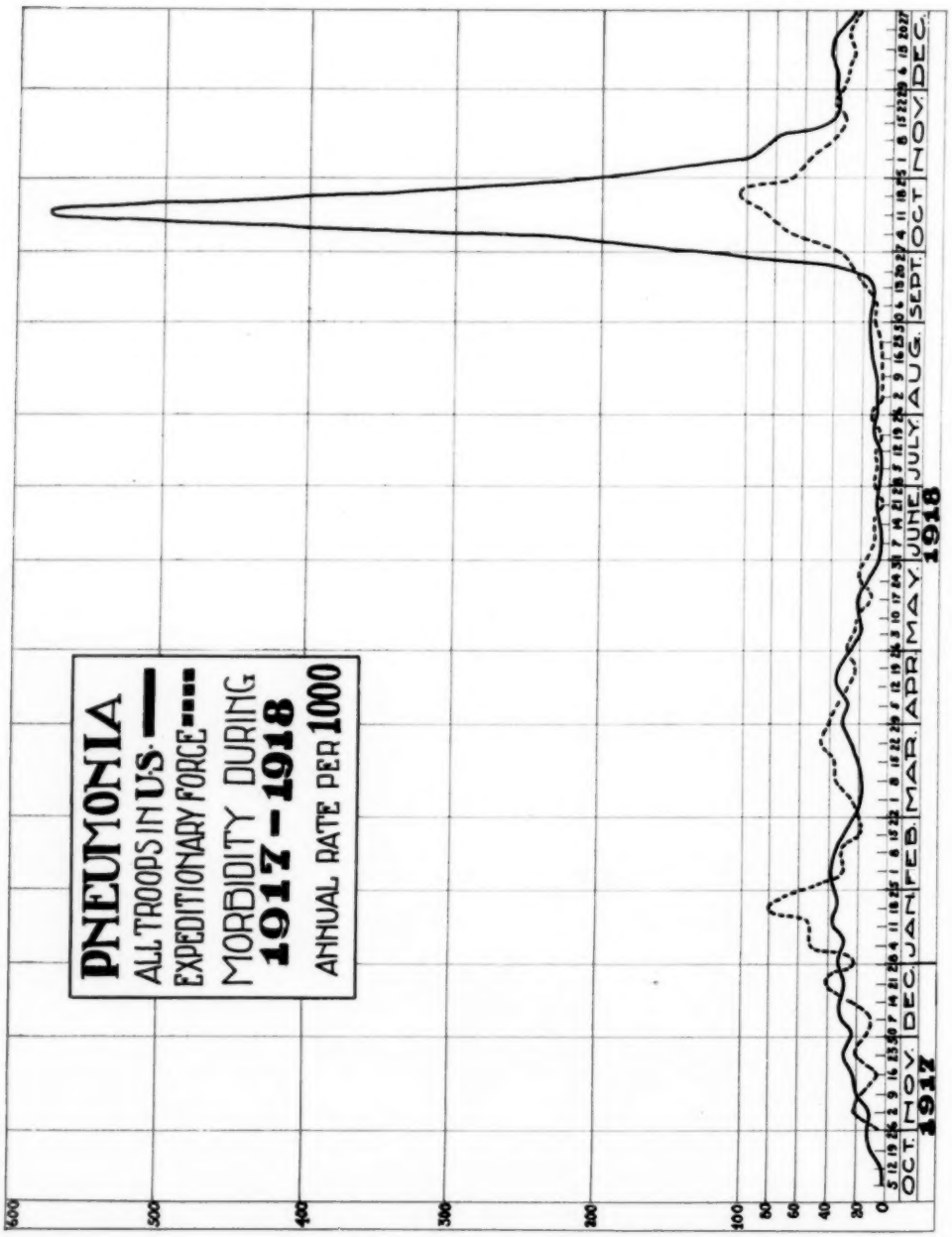


Chart IX.

and trench life, therefore, are of less consequence in making armies free from disease than is the admission of new recruits to the ranks.

Admissions for pneumonia (see Chart IX) began among the troops in this country as early as the third week in October, 1917. From that time until the last week in May the rate fluctuated from 10 to 38, remaining most of the time

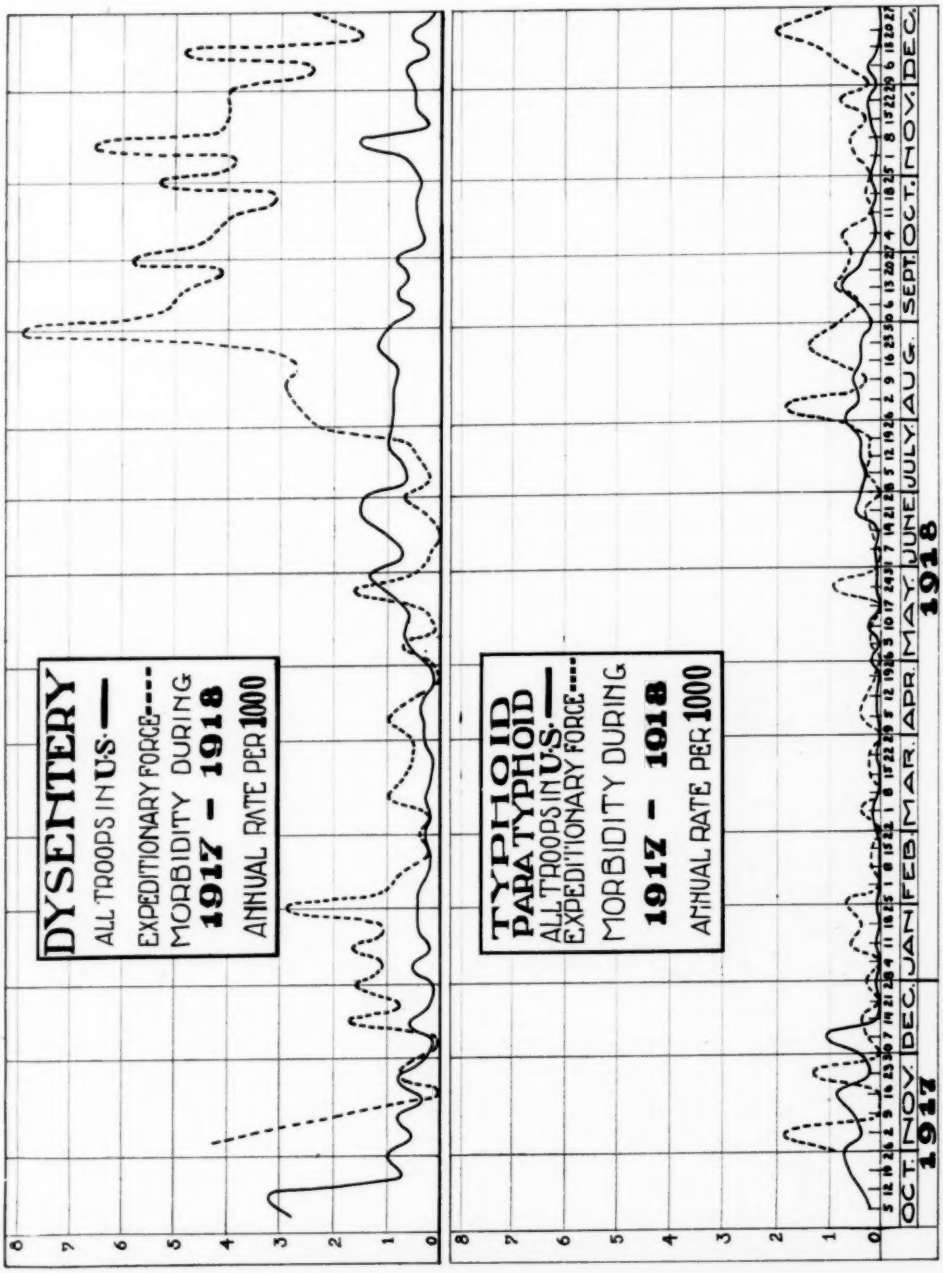


Chart X.

in the twenties and thirties. There was a dropping off after May. During June and July we see relatively little pneumonia. A slight increase occurs during the latter part of August and the figure remains fairly constant up to the third week in September when the influenza epidemic broke out. The highest rate for any week was 592 for the week ending October 11. From that time until

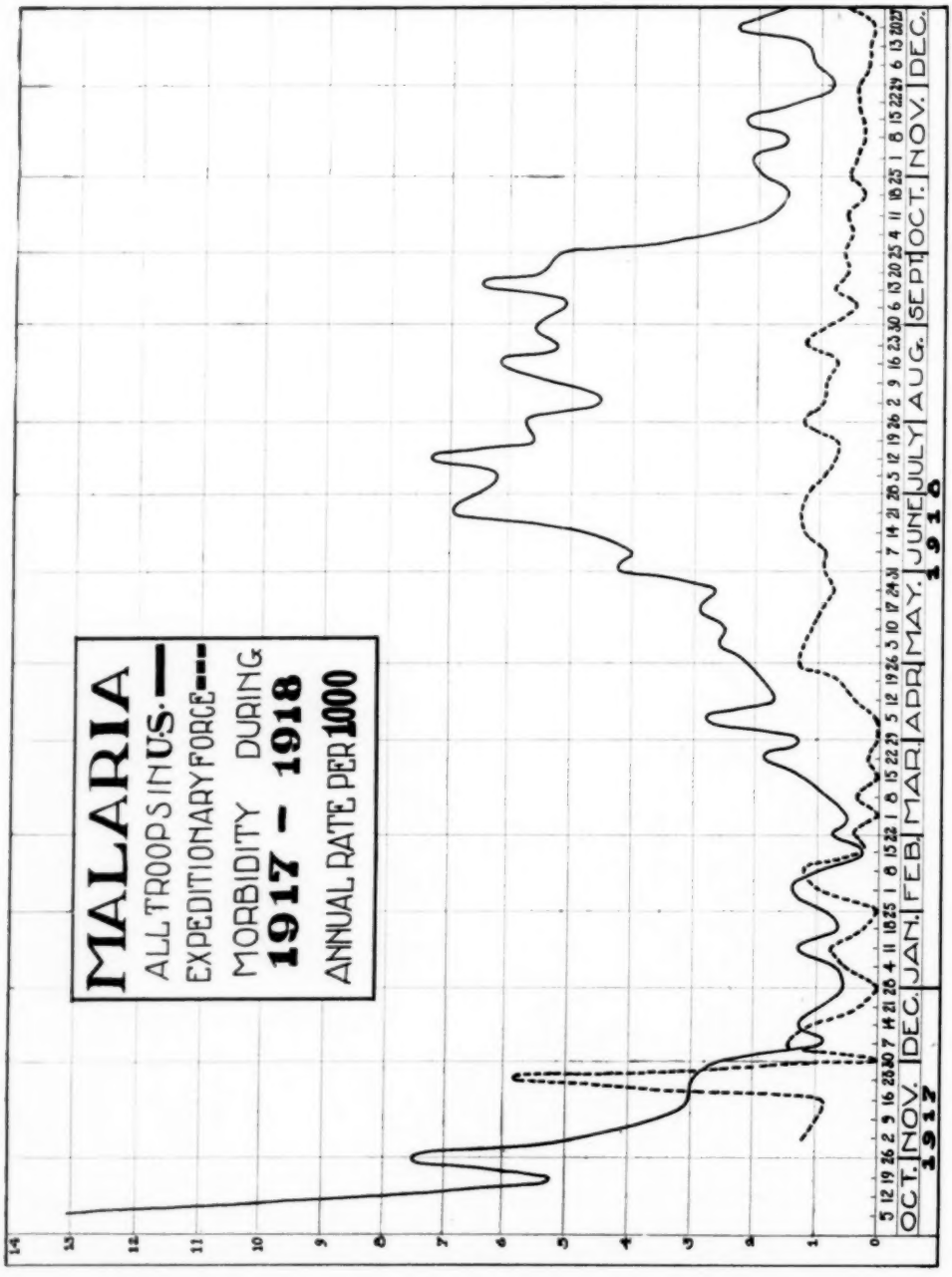


Chart XI.

the end of the year the rate does not fall below 26. Pneumonia in the Expeditionary Force is below that of the troops at home for the first three months. During January the rates for the men abroad are nearly twice as great as the troops in this country. In February the rate is less than for the troops here. From April to October the rates are similar. October and the following months witnessed much higher rates among the troops in this country.

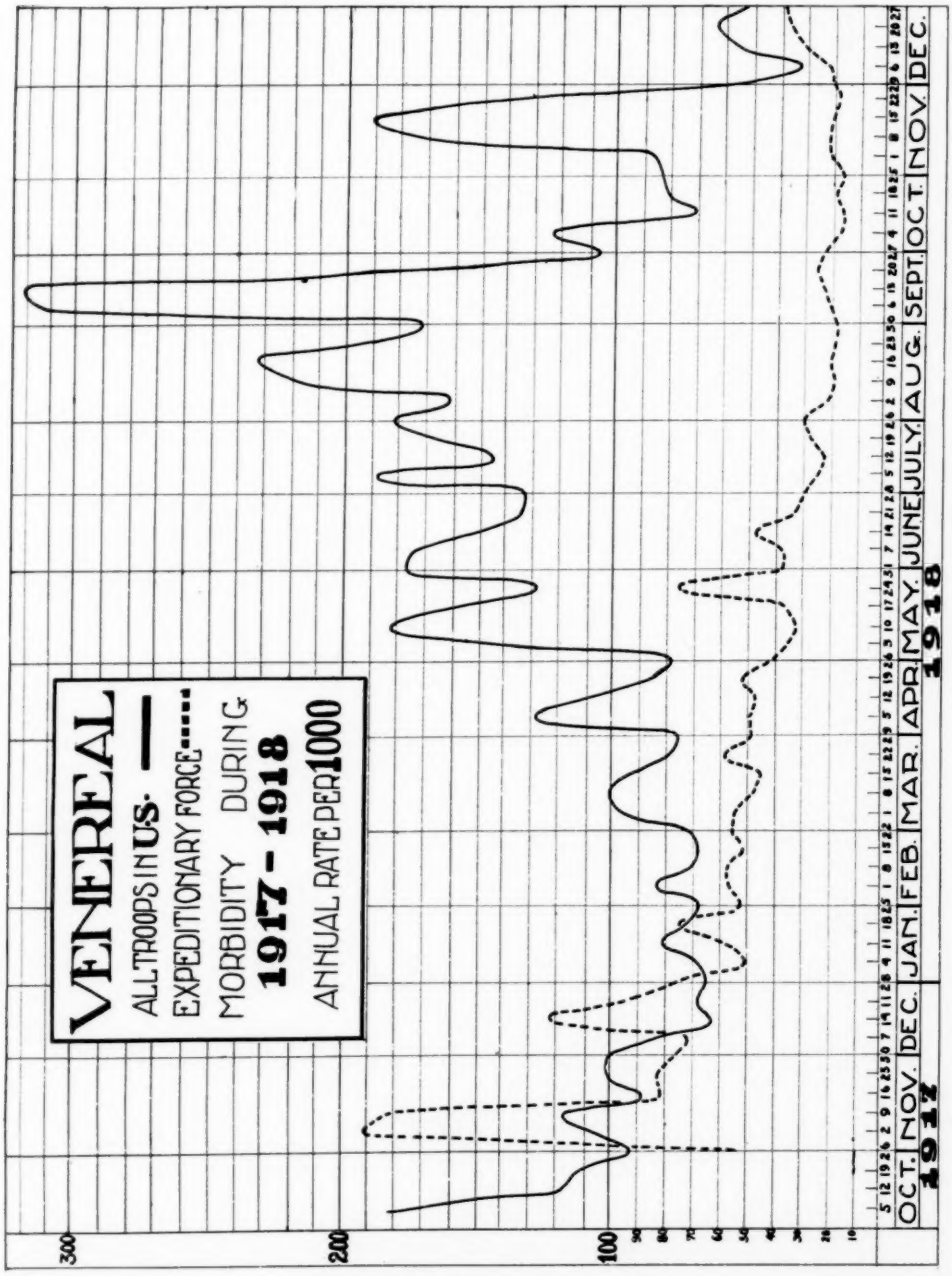


Chart XII.

Dysentery occurred to a very slight degree in this country and abroad. (See Chart X). For the better part of the time, from October, 1917, up to August, 1918, the rates in both usually were less than 1 per 1000. From August however, for the balance of the year the rate for the Expeditionary Force crept up to 2, 3, and even 7.9 for one week, the prevailing figure being around 4. During the same time in this country the rate was always below 1. Differences

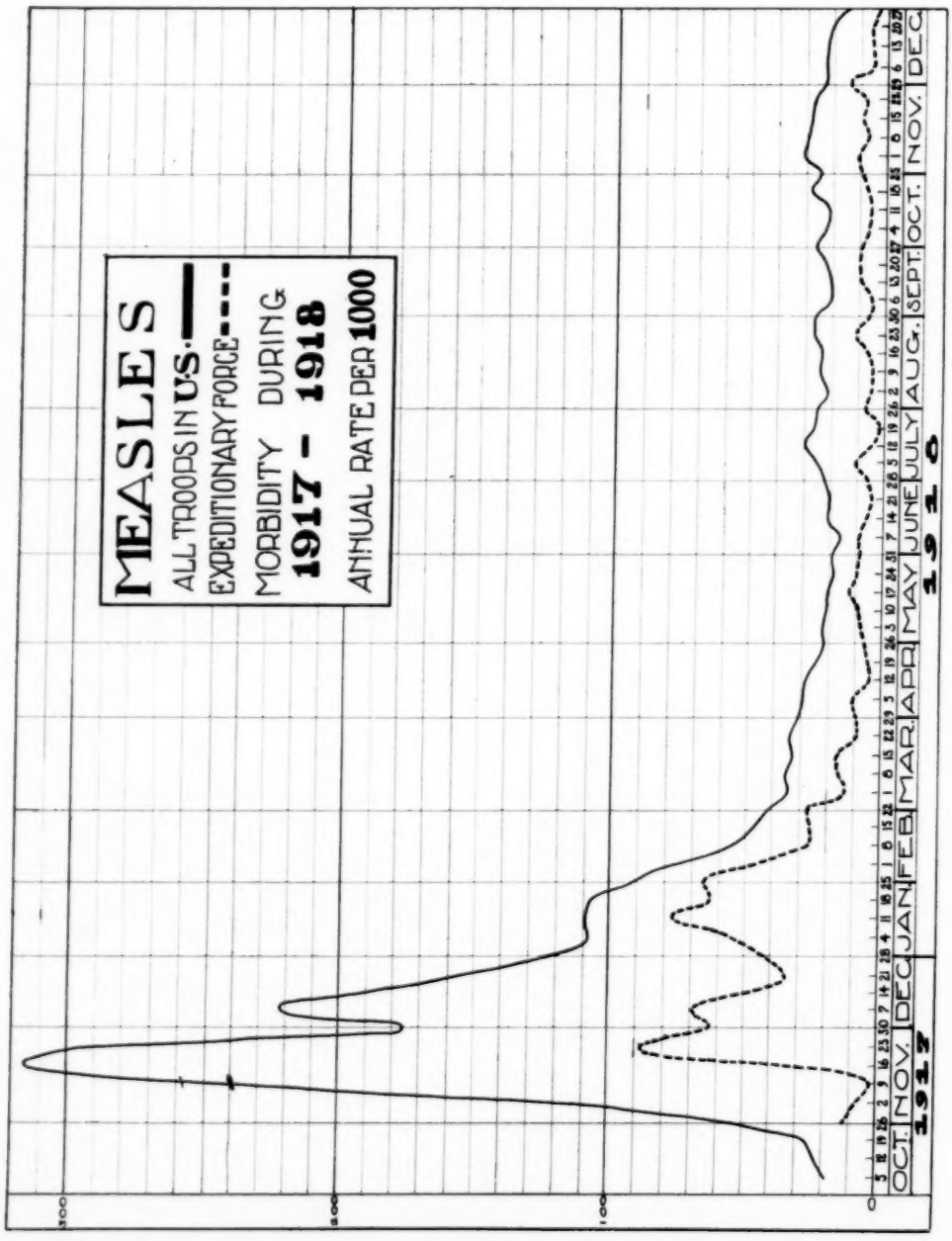


Chart XIII.

in the sanitation of the two forces are thus reflected in the dysentery incidence.

The typhoid rates were less than dysentery. (See Chart X.) On only one occasion during this entire period did the rate for the troops in this country exceed 1 per 1000, and on only seven weeks was the rate for the Expeditionary Force above 1. The highest rate for any week was 2.1, that being the third week in December, 1918. Generally speaking, the rate for the Expeditionary Force was slightly greater than for the troops in this country.

Throughout the entire fifteen months which we are discussing, malaria

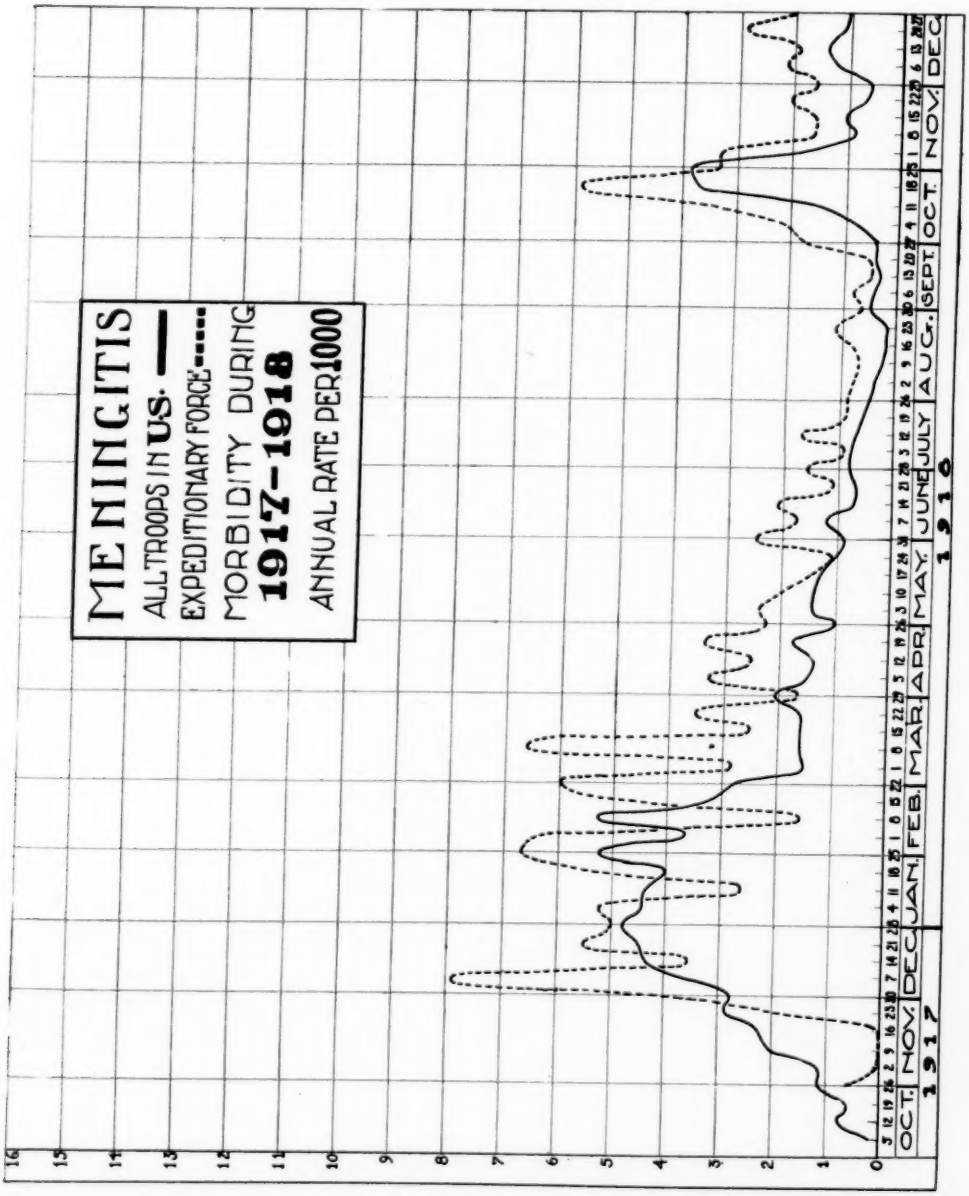

MENINGITIS
ALL TROOPS IN U.S. ——
EXPEDITIONARY FORCE ·····
MORBIDITY DURING
1917-1918
ANNUAL RATE PER 1000
OCT. NOV. DEC. JAN. FEB. MAR. APR. MAY. JUNE JULY AUG. SEPT. OCT. NOV. DEC
1917
1918
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16

Chart XIV.

prevailed to a greater extent among troops in this country than among those abroad. (See Chart XI.) The highest rate was for the first week of this period, that ending October 5, 1917. The rate fell after November to 1 or 2 and continued until May. We are speaking of the home troops. From June till September the rate grew to 5, 6 and 7 and then returned to 1 and 2 for the re-

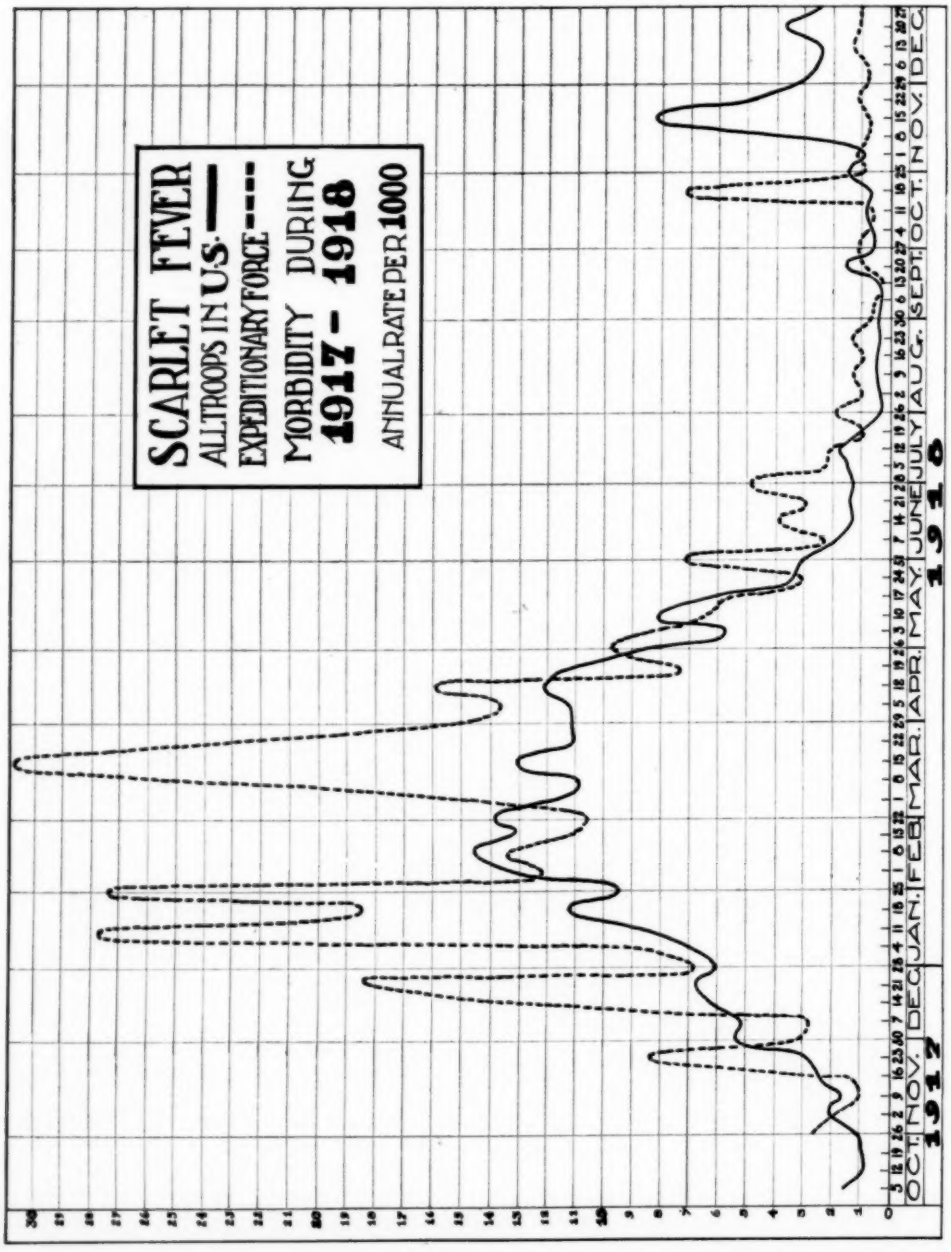

SCARLET FEVER
ALL TROOPS IN U.S. ——
EXPEDITIONARY FORCE ----
MORBIDITY DURING
1917 - 1918
ANNUAL RATE PER 1000
OCT. NOV. DEC. JAN. FEB. MAR. APR. MAY. JUNE JULY AUG. SEPT. OCT. NOV. DEC.
1917
1918

Chart XV.

maining three months of the year. For the expeditionary troops the rate was less than 1 per 1000 two-thirds of the time.

The greatest difference between the two forces will be found in the rate for venereal disease; the home troops having a rate many times that of the force abroad. This is due to the fact that a very great portion of venereal disease occurs among recruits who are infected before enlistment. In a general way the additions of new men to the army are reflected in the venereal rate. This is evident in Chart XII, where we have a high rate during the fall of 1917, the camps at that time being first established. The rate then falls until the spring when new increments are added and the rate rises. The rate continues at a high level until August and September, when new draft calls are made with a corresponding rise in the venereal rate. As the influx of new men ceases in the fall of 1918, the venereal rate drops.

The influence of the new recruit is illustrated also by the weekly rates for measles. (See Chart XIII.) Throughout the entire time the troops in this country have many times the measles admissions of the troops abroad. Apparently there was but one epidemic period, that being in the fall of 1917. From March to December the measles rate was below 40. The rate for the troops abroad was likewise higher during this early period, and after this it fluctuated between 5 and 15.

Meningitis is a disease that prevailed to a greater extent among troops abroad than among troops at home, and this relationship persisted continuously throughout the fifteen months. (See Chart XIV.) There are no great differences between the rates, but that for the Expeditionary Force is always slightly in the lead. The highest rates are from December, 1917, to March, 1918, inclusive, and in October, 1918.

Both the Expeditionary Force and the troops in this country had scarlet fever about the same time; namely, during the first four months of 1918. (See Chart XV.) The Expeditionary Force seemed to suffer more in this respect than the troops in this country, and as is the case with meningitis, the relationship is fairly constant throughout the period, or at least until November, 1918. During November and December the rates in this country are higher than those abroad.

Summing up the experiences in the two armies, that abroad and that in this country, we see first that disease is more prevalent here. The exceptions to this are dysentery, meningitis and scarlet fever. There is more disease in this country because the men are more susceptible. The home army is more susceptible because it contains a greater proportion of new men. The new, unseasoned recruit is therefore responsible for more sickness than is caused among seasoned men by undergoing the hardships of physical and mental fatigue and exposure of combat. In other words, the natural susceptibility of the individual is a greater factor in the causation of disease than environment.

A PHYSIOLOGIC INVESTIGATION INTO THE DYNAMIC ACTION OF THE HEART IN FUNCTIONAL CARDIAC DISORDERS*

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PROBABLY one of the most remarkable facts connected with cases of functional cardiac disease, as encountered in military service, is that no definite abnormality of the cardiovascular apparatus can be found to account for the "effort syndrome." The results of physical examination are either entirely negative or at most bring out a systolic murmur of doubtful import or a slight increase in the area of dullness. Modern instrumental means have also not contributed anything to elucidate the condition; graphic methods have shown nothing of importance; blood pressure studies have yielded only uncontrollable data upon which to build fanciful hypotheses. The electrocardiographic studies so far undertaken indicate that the impulse spreads in a normal manner and rate across the conducting system of the heart—this, it may be added, we are able to confirm.

Nevertheless, the impression can not be eradicated that, when a patient presents such ostensible symptoms—at times amounting to extreme distress—as are designated by the term "effort syndrome," some distinct and tangible abnormality of the cardiovascular apparatus must exist. On *a priori* grounds, one naturally suspects the contractile function of the ventricular myocardium. How do the cardiodynamics of normal individuals and of those with functional disorders compare? This is a question that merits every investigation that modern methods and apparatus can suggest. As a prelude toward such a study, we determined to investigate the duration of the isometric period and the length of systole in those cases upon the basis of considerations which must be briefly presented.

For a number of years, Frank, Tigerstedt, Piper, Starling and the writer[1] have been engaged in studying the dynamics of the ventricle by means of optical manometers recording with a high degree of precision. Such studies have shown that when the ventricles begin to contract, the pressure within the cavities is immediately elevated. This quickly completes the closure of the a-v valves already begun toward the end of auricular systole (Henderson and Johnson)[2] (Dean).[3] As the semilunar valves remain closed while the pressure continues to rise for another .03 to .04 second, the ventricle contracts isometrically, i. e., the ventricular musculature is unable to shorten because no blood is expelled from the cavities and therefore all the energy developed is expended in raising the intraventricular pressure. To this period of rising tension, one of us has given the name "isometric period." Upon opening of the semilunar valves, blood is

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ejected and the ventricular fibers shorten. This is the "ejection period" or the "auxotonic period" of the heart.

Experimental evidence has shown in a fairly satisfactory manner that whenever the contractile power of the ventricles is impaired, the pressure rises less rapidly within the cavities during the isometric period and, consequently, this period is lengthened. This being so, the period of systole is lengthened. It is therefore evident from such experimental work that the impaired ventricle differs from the normal in having a longer isometric period and consequently an increased duration of systole.

A similar, but more general conception has been suggested by Lombard and Cope,[4] who, impressed by the fact that the period of contraction is lengthened in fatigued skeletal muscle, conceive it possible that the fatigued cardiac muscle may similarly show a prolonged systole. According to this view, the systole in the functionally impaired heart would not be terminated as early as that of the normal heart, such systole being longer without any necessary lengthening of the isometric period.

Is either one of these deviations perhaps present in the case of functional cardiac disorders? To throw light on this question, we compared the duration of the isometric periods and the entire systolic period of functional cardiac cases with those of normal subjects and with organic heart cases.

METHODS OF STUDY

An accurate study of the isometric period and duration of systole requires a most reliable means for their determination in man. Frank has pointed out that the isometric period is not a presphygmic event as has long been taught, but is marked as a small preliminary wave on the carotid pulse curve, when accurately recorded by optical capsules. In clear-cut records the duration of this period may therefore be directly determined. The end of systole is also clearly indicated by a sharp incisura so that the entire interval of systole may thus be directly calculated.[5] It was soon found, however, in routine examination of patients that many tracings were obtained in which the beginning of the preliminary wave was in doubt, especially when, as sometimes happened, a double vibration preceded the primary rise of pressure. It was therefore necessary to record simultaneously the heart sounds which was conveniently done by the improved sound recording capsules described by one of us and Dean.[6]

Technical.—In order to determine with precision the onset of the isometric period, a little construction is necessary, as the following considerations will show:

The main vibrations of the first sound coincide with the beginning of the ventricular pressure rise and therefore mark the onset of the isometric period. The vibrations of the second sound fall a short interval after the end of systole and correspond with the bottom of the incisura of the pressure curve in the aorta. To correct accurately for delay in transmission of vibrations to the neck, it is necessary therefore to place a pair of dividers with one point on the onset of the first sound vibrations and the other on the second sound vibrations and transfer the set dividers to the carotid curve so that one point is placed at the bottom of the incisura. The other point then cuts the wave representing the beginning of the isometric period. The interval between this mark and the rise of the primary wave gives the isometric period, while the distance from this mark to the beginning of the incisura gives the duration of the total systole. It is obvious that the correct interval for systole so determined is slightly shorter than the interval between the onset of the two heart sounds.

Relation between Duration of Systole and Cycles of Different Length.—It has been a generalization in physiologic teaching that any change in the duration of the cardiac cycle occurs at the expense of diastole, the period of systole being unaffected. Careful studies have shown that, while the major change occurs in diastole, systole is also affected. This variation is so pronounced that it actually affects the duration of systole from one heartbeat to another. This has been recently studied in great detail by Lombard and Cope[4] who found it necessary to establish an average period of systole for every length of cardiac cycle. Inasmuch as their data were published only in preliminary form and were, moreover, obtained by tambours recording on a smoked surface, therefore not taking into account the isometric period, it was necessary to first study a similar series of normal cases with which to compare the functional cases of cardiac disorder. To these classes of individuals studied was added a third of undoubted but well-compensated organic valvular disease. The cases of each group were carefully selected by Major F. Peabody and each case was placed in its group only after careful examinations and conferences between Major F. Peabody and the members of his staff. Since Lombard and Cope have recently[7] found considerable variation in systole between the standing and sitting posture, it is desirable to add that all observations were taken in the sitting position.

RESULTS

A. Normal Subjects.—Normal subjects were selected from the military service, being largely officers who had undergone careful physical examination. The results were first tabulated after the following scheme: After each subject in a column headed "cycle," the duration of the longest and shortest cycle was recorded. The figures for the duration of the isometric period were then extended in a column corresponding to the duration of systole. In this way the duration of the isometric period and of systole was conveniently tabulated for cross reference. Thus, figures for cycles ranging from 1.07 sec. to .59 sec. (corresponding to heart rates of from 56 to 100 per minute) were available. As the data in regard to heart rates above 80 per minute were rather scant and no rates above 100 per minute were encountered during rest, some of these subjects were requested to exercise by hopping one hundred times on one foot and the records were again taken as soon thereafter as possible. The data were similarly collected and tabulated. In order to economize space, these and similar tables are not now published but the combined data regarding the duration of systole were plotted in a common chart shown in Fig. 1. In this and subsequent plots, the dots represent the data obtained during rest, and the crosses, the data obtained after exercise.

Results.—The following results may be briefly emphasized:

1. Of 24 cases studied, 6 (25 per cent) had a pulse rate over 80 while resting.

2. The duration of the isometric period in man is somewhat variable and bears no definite relation either to the heart rate or the duration of systole. The extreme ranges were from .025 sec. to .08 sec. although the largest number of cases came within the range of .04 - .06 sec.

3. The general trend of the plotted curves, shows that the duration of sys-

tole becomes progressively greater as the cardiac cycle increases. *In general, it may be said that the duration of systole in normal men is below .25 sec. when the heart rate is above 100 per minute; averaging about .25 sec. when the heart ranges from 80 to 100 per minute, and about .28 to .30 sec. when the heart rate is between 66 and 80 per minute, while it is distinctly longer than .30 sec. when the heart is slower than 66 per minute.*

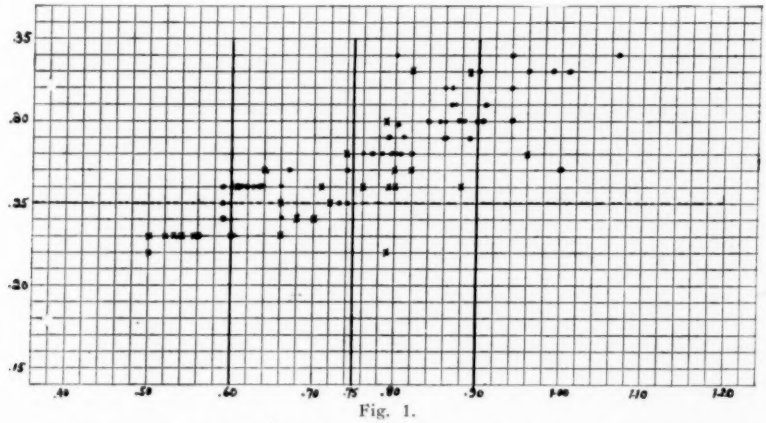

Fig. 1.

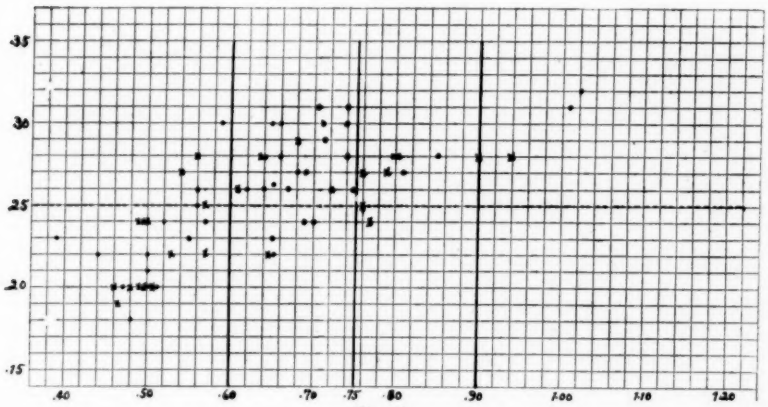

Fig. 2.

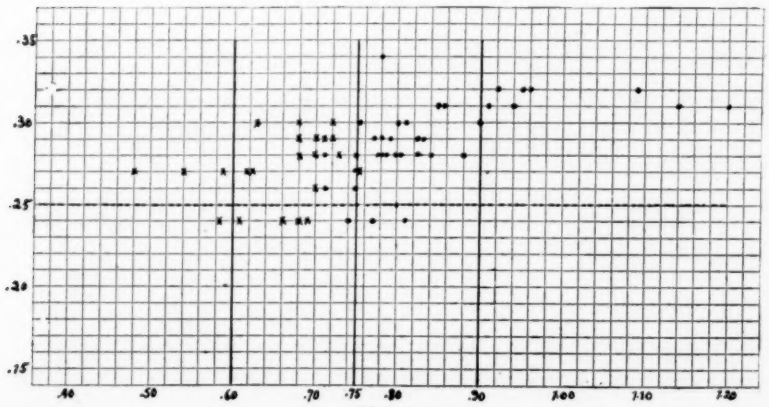

Fig. 3.

B. Functional Cardiac Cases.—The results obtained from functional cardiac cases during rest and exercise were similarly tabulated and from these the plot shown in Fig. 2 was made. The results may be briefly summarized:

1. Out of 25 cases examined, 21 (84 per cent) had a pulse rate above 80 per minute during rest. This confirms the general clinical observation that cases of functional cardiac disorders have a much more rapid heart rate than normal subjects.

2. As in normal hearts, the duration of the isometric period with few exceptions falls within the range of .04 to .06 sec. and bears no definite relation to the duration of systole. *It is clear that these hearts do not show any demonstrable deviation similar to the depressed myocardium of experimental animals in which the duration of this period is lengthened.*

3. Owing to the fact that the largest number of normal hearts have a rate below 80 per minute but can be accelerated above this rate by exercise, and by far the largest number of functional cases have rates above 80 per minute even at rest, we are justified in comparing only the data within the range of 80-100 per minute, for in this range alone are the observations sufficiently numerous and equally balanced to be really reliable. Doing this, we find that when the cycles range from .60 to .75 sec. the phases of systole are distinctly above .25 sec., the average length in normal hearts. This, it may be emphasized, is a distinct difference between normal hearts and the hearts of functional cardiac cases.

4. While the duration of systole for cycles of corresponding length, i.e., between .60 and .75 sec. is distinctly longer than in normal hearts, it is evident that the power of shortening the systolic phase is not lost. The periods plotted in Fig. 2 for cycles longer than .60 sec. indicate clearly that the duration of systole may be reduced as the heart becomes very rapid. Unfortunately not enough normal data on such high heart rate were obtainable for comparison.

C. Compensated Organic Cardiac Cases.—The organic cardiac cases studied represented a miscellaneous group of mitral and aortic lesions in which auricular action was regular and no sign of decompensation was present. These results were again tabulated and are plotted in Fig. 3. The essential features to be emphasized are:

1. Of 33 cases studied, 12 (36 per cent) had a heart rate over 80 and of these only 3 (9 per cent) had a rate over 100 per minute. This is in agreement with clinical observations that organic heart disease (uncomplicated by auricular fibrillation) is not characterized by rapid heart action.

2. The isometric period did not deviate from the normal range, showing that in compensated hearts considerable valvular derangement may exist without an alteration of the myocardial function similar to that found in experimental animals.

3. If we compare these groups of cases having before and after exercise a heart rate between 80 and 100 per minute with similar cases of normal and functional hearts, we find that there is a greater variation in the duration of systole than shown in the other groups. This is, no doubt, attributable to the varied character of the organic disorder which it was not our aim to investigate. It is evident, however, that, as in functional cardiac disorders, a large number of cases have a systolic phase definitely above .25, thereby indicating a prolongation of systole.

4. When we similarly compare the range of systole in organic cases having a normal heart rate (66 to 80 per minute) with that of normal hearts, little difference is found. In other words, we may say that hearts organically diseased when beating at the normal rate, have a systole of normal length which fails to shorten, however, as the heart accelerates.

CONCLUSION

A Possible Physiologic Conception as to the Nature of the Derangement in Functional and Organic Cardiac Disease.—A consideration of these data in the light of physiologic facts makes possible the following conception as to the cardiac derangement in cases of functional cardiac disorders and at least certain types of compensated organic lesions:

The normal heart possesses a mechanism whereby the systole is shortened when the rate is accelerated. This is especially evident when the rate increases beyond 80 per minute. In this way, we may suppose the musculature, while subjected to more frequent periods of contractile stress, is automatically spared to some extent through a shortening of systole. In functional cardiac disorders, this compensatory mechanism (whatever its nature and cause) may be entirely abrogated, or, what is more common, does not become operative until the heart has accelerated to a far greater extent (e. g., to 100 to 120 per minute). In other words, whenever the functionally disordered heart is accelerated above 80 per minute, its period of systole is not shortened as that of the normal heart and, consequently, the myocardium is exposed more frequently to a prolonged period of contractile stress. This condition, favoring the onset of rapid cardiac fatigue is more serious since the hearts of these subjects are usually rapid even at rest.

It appears that the same deficiency in the adjustment of systolic length to the duration of the cardiac cycle exists in many organic heart cases. Owing to the fact, however, that the heart rate in these individuals is usually slow, at least during rest and moderate activity, the cardiac myocardium is in this way spared from the consequences of such failure to shorten.

In conclusion, it may be stated that this report represents but a preliminary study of this question. Whether this dynamic alteration will be found characteristic of larger groups of cases, only a more prolonged study of such cases can determine. Whether the cardiac fatigue produced by such a deviation in the dynamic action of the heart is sufficient to account for the "effort syndrome" must prove the subject for an experimental investigation in the future.

We wish to thank Mr. Fred Keyes and Corporal Ford for their technical assistance in carrying on this investigation.

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NOTES ON THE EFFECT OF INTRAVENOUS DIARSENOL*

By W. C. Rappleye, M.D., Foxborough, Mass.

I. THE EFFECT ON RED CELL FRAGILITY

IT has been reported by several observers that administration of arsenic by mouth in different types of anemia has resulted in a distinct increase in the resistance of the red corpuscles to hypotonic salt solution. Gunn, in 1908, observed that the immersion of red cells for an hour in a 1:10,000 solution of arsenious acid increased the resistance of the cells to hypotonic saline and also showed later that the increased resistance conferred by arsenical substances was operative against hemolysis by hemolytic chemical substances. Hill was able to demonstrate the antihemolytic action of arsenic in chemical tests. In a case of "pernicious anemia," administration of Fowler's solution for ten days in increasing doses distinctly increased the resistance of the red cells to hemolysis by hypotonic salt solution. Before treatment, the beginning of hemolysis occurred in 0.475 per cent saline and complete hemolysis took place at 0.325 per cent, whereas the corresponding strengths of solution causing hemolysis after treatment were 0.450 and 0.275 per cent.

Similar studies have been made with the organic arsenical compounds, salvarsan and neosalvarsan. It has been found that *in vitro*, the presence of salvarsan delays hemolysis. In a very small number of clinical cases, red cell fragility has been determined before and after injection of salvarsan (0.3 gm. intravenously) and no change in the zone of hemolysis could be made out. In the treatment of a few cases of the Addison-Biermer type of hemolytic anemia, however, the resistance of the red cells to hypotonic salt solution was definitely increased by intravenous salvarsan therapy. Whether the decreased fragility is due to an increase in the resistance of the existing red cells or due to stimulation of the bone marrow and production of young red cells which, according to Snapper and others, are more resistant, is not at once apparent. The leucocytosis noted after intravenous salvarsan treatment may be some evidence in favor of the latter possibility.

During the diarsenol treatment of cases of general paresis in the hospital here, a study of the red cell fragility was made on the blood of these patients. Determinations of the zone of hemolysis were made on the blood before intravenous treatment and at intervals afterward. It must be borne in mind that the blood of these patients was essentially normal in so far as nothing of note in the cytology, hemoglobin content or blood counts was found. For this reason, one might be inclined to expect no change in the fragility determinations following the introduction of diarsenol into the blood stream.

The dose of diarsenol used in each instance was 0.5-0.6 gm. (8.0-9.0 gr.), representing 0.15-0.18 gm. (2.4-2.7 gr.) elemental arsenic, a considerable amount of arsenic were it not for the fact that it is in organic combination and 50-75 per cent of the diarsenol is promptly excreted unchanged. The further excre-

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tion of arsenic into the urine in some other form (as yet undetermined) and the rapid decrease in the amount found in the blood plasma gives but a limited opportunity for the arsenic to act upon the red cells, although it seems established that arsenic is found in the red corpuscles for some time after the plasma is free from it. The figures are given in Table I.

TABLE I

CASE	ZONE OF HEMOLYSIS BEFORE TREATMENT %	TIME AFTER TREATMENT	ZONE OF HEMOLYSIS AFTER TREATMENT %
1	0.44 to 0.31	5 min.	0.43 to 0.28
2	0.42 to 0.27	5 "	0.44 to 0.29
3	0.46 to 0.30	5 "	0.46 to 0.30
4	0.42 to 0.30	5 "	0.44 to 0.30
5	0.42 to 0.32	5 "	0.44 to 0.31
6	0.42 to 0.28	10 "	0.43 to 0.30
7	0.40 to 0.28	10 "	0.40 to 0.29
8	0.42 to 0.31	10 "	0.43 to 0.31
9	0.44 to 0.34	30 "	0.44 to 0.34
10	0.44 to 0.32	30 "	0.43 to 0.32
11	0.44 to 0.33	30 "	0.44 to 0.33
12	0.45 to 0.29	1 hour	0.46 to 0.30
13	0.46 to 0.32	1 "	0.45 to 0.33
14	0.44 to 0.33	1 "	0.44 to 0.33
15	0.46 to 0.33	1 "	0.45 to 0.33
16	0.45 to 0.28	3 hours	0.45 to 0.30
17	0.44 to 0.31	3 "	0.45 to 0.32
18	0.46 to 0.33	24 "	0.45 to 0.34
19	0.44 to 0.31	24 "	0.45 to 0.33

It seems quite evident from these figures that the fragility of normal red cells is not altered by diarsenol introduced directly into the blood stream. Such a conclusion, however, does not warrant a statement that in the abnormal blood conditions, such as the hemolytic anemias, the introduction of arsenical compounds into the blood stream does not increase the resistance of the red cells.

II. THE EFFECT ON BLOOD UREA NITROGEN

It is known that the administration of arsenic in inorganic form, as long as the substance does not interfere with digestion or absorption, increases the excretion of nitrogen, the total nitrogen of the urine being increased, especially the urea and ammonia fractions. Presumably this is caused by a mutual interaction of several factors, including capillary dilatation, a direct action on the body cells, and an action upon the gastrointestinal tract and kidneys. Weiske, however, has noted a decrease in nitrogenous excretion and especially in the amount of nitrogen in the stool and it has been maintained that the improvement in nutrition following the administration of arsenic is due to more complete utilization of nitrogen by the digestive tract and a decreased decomposition of protein by the tissues. The variations in the elimination of nitrogen, however, vary within narrow limits and possibly are not related to the action of arsenic at all. The substance disappears rapidly from the blood, being excreted or taken up by the tissues, presumably forming firm compounds with the nucleins.

Not much literature is available dealing with the effects of salvarsan or diarsenol upon metabolism. During the treatment of cases of general paresis here with intravenous diarsenol, a few observations were made upon the blood urea nitrogen values and their behavior in relation to the introduction of the organic arsenic. As commented on in the note on red cell fragility, the diarsenol given intravenously disappears rapidly from the blood stream, the greater part of it being excreted promptly. And since the balance is in organic form or probably combined with the nucleins or other body substances, the amount of arsenic available to influence metabolism must be small.

The plan followed was to draw the blood just before giving the intravenous diarsenol, i. e., secure a sample of blood from a needle already in place for the intravenous therapy, and then to secure samples at intervals after the injection, one hour, three hours, and twenty-four hours after treatment. The determinations were made by the Marshall urease method, using the aeration and titration procedure. Each determination was made on 5 c.c. of blood.

Control of the various factors influencing the blood urea values, even the fairly well-understood factors, is difficult. The influence of diet, water intake, and other features in the day's routine calls for consideration and should be controlled in order that any variations in the urea nitrogen values ascribed to the dye may be appraised with more accuracy. Accordingly, patients were given the diarsenol before breakfast, the control blood having been taken previously. On different occasions, blood was again drawn at the end of one hour, three hours, and twenty-four hours after administration, the patient having taken no food or water during the short intervals. The close approximations of the blood urea nitrogen values on the same individuals on blood drawn before breakfast at intervals of several days, when there is little or no kidney impairment, the individual remaining, of course, on a similar diet and routine, is well known. So the values obtained twenty-four hours after the diarsenol administration are relatively uninfluenced by the diet of the previous day, the blood being again drawn before breakfast.

The blood urea nitrogen values will be dependent in part on the functional efficiency of the kidneys. And the influence of diarsenol upon these values will probably vary then with the kidney function. If the diarsenol acts in any way as a renal irritant, it may be expected that the urea excretion will be accentuated in the presence of kidneys which will respond readily to the irritation. Still, our knowledge of the action of arsenical compounds upon metabolism is as yet inadequate to allow us to be sure that such action does not alter the nitrogenous substances of the blood quite independently of any renal action. The rate of phenolsulphonephthalein excretion was ascertained in most of the cases here and it can be said that, in this group of patients, the decrease or increase in blood urea nitrogen following the use of diarsenol does not seem to bear any relationship to the dye elimination. The figures are shown in Table II.

In the group of cases noted in Table II, it will be seen that the introduction of diarsenol into the blood stream affects the urea values but little, if any. The variations are within normal range. It is true that there is a slight majority of the cases which showed a drop rather than a rise in values durjng the first few hours after the injection. But the drop is not conclusive, surely not as striking as the

TABLE II

CASE	BLOOD UREA NITROGEN BEFORE TREATMENT	TIME AFTER TREATMENT	BLOOD UREA NITROGEN AFTER TREATMENT
1	12.9 mg. -100 c.c.	1 hour	11.8 mg.-100 c.c.
2	7.8	1 "	8.4
3	10.1	1 "	7.8
4	8.4	1 "	9.5
5	11.2	1 "	10.1
6	8.9	1 "	7.8
7	13.1	1 "	15.6
8	15.4	1 "	18.5
9	10.1	1 "	13.1
10	17.6	3 hours	16.0
11	15.4	3 "	16.5
12	9.2	3 "	8.4
13	11.7	3 "	9.2
14	14.0	3 "	13.4
15	12.3	3 "	11.7
16	13.2	24 "	11.2
17	14.0	24 "	13.7
18	8.7	24 "	10.1
19	16.0	24 "	15.4
20	9.6	24 "	11.2

drop in blood urea nitrogen noted in catatonia and reported in another connection.

III. THE EFFECT ON KIDNEY FUNCTION

The danger attending the use of salvarsan or diarsenol therapy in cases of nephritis of a nonsyphilitic character is definite. It is known that most of the diarsenol is rapidly excreted from the blood into the urine, at least when the functional efficiency of the kidneys is good. The drug remaining in the body apparently combines with the nucleins. It is not clear whether the adverse action in nephritis is due to direct action on the kidneys by the circulating drug and the attendant attempt at its excretion or is less direct. Since in such cases the excretory function of the kidneys is depressed, it may mean that only a small portion of the diarsenol is excreted as such, leaving the greater part in the body where it is gradually altered in character or combined with the cell proteins; or the portion in circulation may yield arsenical substances which compromise more completely a deficient kidney function. The relationship of kidney function and the excretion of such substances as salvarsan has been but incompletely investigated. The fact that salvarsan is a dye substance and that we judge kidney function in part by the rate of elimination of easily demonstrable dye substances such as phenolsulphonephthalein, does suggest that in depressed kidney function there is an unusual retention of diarsenol after its use, which may account for the difficulties seen in cases of nephritis in which it is used, rather than a direct action of the drug on the kidneys.

The observations made here throw no light on this feature, for the diarsenol in the urine was not followed. The point of interest was to learn whether, after the use of considerable amounts of diarsenol, there could be demonstrated evidences of renal insufficiency which could be ascribed to the drug itself. The number of cases for study was small and for that reason warrants no generalization. But it is reasonable to expect that in cases which have received many treatments

of diarsenol over a long period there should be some evidence of nephritic impairment if diarsenol itself is a kidney irritant of any severity. It is well recognized that kidney impairment is distinct in late stages of neurosyphilis and the kidney insufficiency seen in cases of general paresis which have been actively treated with salvarsan, for example, should not be ascribed to the therapy unless we can conclude that the degree or frequency of impaired excretory function is greater in treated than in untreated cases of neurosyphilis. If organic arsenical compounds are excreted without special irritation to the kidney, we can expect the renal function to be but little altered even after many injections. It seems likely, too, that the untoward effects of organic arsenical compounds in cases of nonspecific nephritis is due more to the breakdown of the compound in the body, the kidneys possibly failing to eliminate this dye as they also fail to excrete phenolsulphonephthalein, than to direct renal action. That the renal phenomena may be due to the results of the breakdown must be granted.

The rate of elimination of phenolsulphonephthalein, the blood urea nitrogen, blood pressure and urine analysis were done on a small group of cases that had received a considerable amount of diarsenol treatment. The doses had ranged from 0.3 gm. to 0.6 gm.

The figures for this group are given in Table III.

TABLE III

CASE	DIARSENOL	DYE %	BLOOD UREA NITROGEN	BLOOD PRESSURE	SP. GR.	URINE ALB.	URINE CASTS
*G. B.	19.6 gm.	20	8.9 mg. 7.8	120-85	1.014	S. T.	Hyal. Gran.
*E. L.	24.9	10	18.5	102-70	1.009	0	Occ. Hyal.
J. M.	16.0	60	10.1 13.1	120-85	1.010-18	0	0
F. H.	29.6	60	15.6 13.1	115-70	1.024	S. P. T.	Gran.
M. J.	26.5	45	11.0 9.5	118-65 155-70	1.010	0	0
J. J.	15.6	40	15.2	124-80	1.030	0	0
R. D.	11.0	35	12.4	110-70	1.025	V. S. T.	Gran.
C. D.	32.	60	8.4 11.2	115-80	1.010	0	0
H. F.	12.5	30	15.4	152-85	1.018	S. P. T.	Gran.
M. C.	17.5	50	13.4	115-80	1.016	0	0

*In bed, showed considerable edema, general condition poor.

It will be noted that several of the above patients had received a good deal of diarsenol and so far as clinical pathology shows, their kidney function remains good. We have had a much larger group of cases under treatment and similar studies on them fail to demonstrate that diarsenol has a deleterious effect on the kidneys when their function is good at the outset.

LABORATORY METHODS

AN EDESTIN METHOD FOR THE ESTIMATION OF PEPSIN IN STOMACH CONTENTS*

By Paul R. Farrington, A.B., and Robert C. Lewis, Ph.D., with the cooperation of Philip W. Brown, A.B., Boulder, Colo.

THE purpose of the investigation here reported was to devise a practical, easy, and rapid method for the preparation of a good and uniform quality of protein, which could be substituted for the pea globulin in the method of Rose[1] for the determination of pepsin. On account of the complicated technic involved in the preparation of pea globulin and because of the difficulty of obtaining it commercially, it seemed desirable to bring this admirable method of Rose, as modified by Givens,[2] into the hands of the practitioner by using a protein which could be readily secured. No attempt was made to obtain a chemically pure protein. Our object was rather to get a product of fairly constant composition by as simple a technic as possible.

As shown in preliminary experiments, edestin has all the qualities needed in a substitute for the pea globulin of the Rose method. It is precipitated from sodium chloride solution by the addition of acid, is readily digestible at 50° C., and may be kept in dilute solution without deterioration for several months. Like pea globulin, edestin is nonpoisonous, thus removing one of the objections to the Jacoby-Solms[3] ricin method. Coupled with these essential properties we have found that edestin may be obtained by a relatively simple procedure. The following method of preparation has proved most practical in our hands.

PREPARATION OF THE EDESTIN

One hundred ten grams of finely ground† hempseed meal were shaken in a bottle with a pint of gasoline to extract a portion of the fat, and the yellow, oily gasoline was filtered off through a folded filter. The meal was then transferred to an evaporating-dish and dried by placing it in the sun, or on a radiator, first making sure that the temperature could not get above 60° C. The meal was stirred to break up all lumps and treated in a clean flask with 200 c.c. of a filtered solution of 10 per cent commercial sodium chloride (common salt). A little toluol was added, the mixture was shaken for a half-hour and allowed to stand overnight in a cool place. Next day, the flask was placed in a water-bath at 50° C. and allowed to remain there with frequent shaking until it had attained the temperature of the bath. The mixture was strained through cheesecloth to re-

*From the Henry S. Denison Research Laboratories of the University of Colorado.

†The meal may be ground in a coffee mill or meat chopper.

move the hulls, reheated to 50° C., and filtered through a folded filter in a water-jacketed funnel kept at 50° C., renewing the papers whenever the rate of filtration became too slow. The first few cubic centimeters that came through were reheated to 50° C., and, if any insoluble material was left, the extract was poured back upon the filter. It took about two hours to filter 100 to 125 c.c. from the original 200 c.c. by this method. When this amount of filtrate was obtained, it was reheated to 50° C. to make sure that no insoluble material had gone through the filter. In case a precipitate remained after heating, it was removed by filtration.

The extract was now poured into a half-gallon grape-juice bottle fitted with a two-hole stopper through which two tubes were inserted, one extending about 4 inches through the cork, and the other to the bottom of the bottle. A little toluol was added to prevent putrefaction, and the whole was mixed with 9 volumes of pure water, thus causing the protein to separate out as a white, flocculent precipitate. The short tube was closed with a piece of rubber tubing and a pinchcock, and the bottle, securely corked, was inverted, supported by a clamp and placed in a cool place. In four to eight hours the edestin settled out in the neck of the bottle, and the clear supernatant liquid was drawn off through the shorter tube, which was adjusted to remove the last portions of the clear fluid. The volume of the wet precipitate was measured, 19 volumes of water and a little toluol were added and the bottle was recorked, shaken, and inverted. The precipitate was allowed to settle and the cloudy supernatant liquid was decanted as before. The wet precipitate was placed in a large watch glass, and allowed to stand in a warm place until a dry cake of friable, brown material remained. This was ground in a mortar to a fine, almost white powder.

Four samples of edestin, prepared by the method outlined above, were found to have a surprisingly uniform edestin content. Table I shows the various steps in the preparation of these samples as well as the edestin content of the finished product. Enough edestin was obtained in each sample to carry out at least 500 analyses for peptic activity by the method proposed below.

TABLE I

SHOWING THE VARIOUS STEPS IN THE PREPARATION OF THE EDESTIN, THE PURITY OF THE PRODUCT OBTAINED, AND THE PERCENTAGE OF NaCl PRESENT

SAMPLE NO.	1	2	3	4
Extract, c.c.	123	110	112	118
Water added, 1st. dilution, c.c.	1107	990	1008	1062
Remaining, 1st. decantation, c.c.	100	85	75	93
Water added, 2nd. dilution, c.c.	1800	1615	1425	1767
Remaining, 2nd. decantation, c.c.	75	50	60	68
Edestin obtained (approximate), grams,	4.25	2.75	2.5	3.25
Edestin by Kjeldahl (5.34 × N), %	88.53	89.89	90.94	90.35
Calculated NaCl*, %	0.92	1.0	1.09	1.04

*An attempt to actually determine the chloride in a small portion of edestin (Sample 1, Table I) showed only a trace.

METHOD OF PEPSIN DETERMINATION

Inasmuch as the normal range of peptic activity is comparatively great and slight variations in the pepsin number have no clinical significance, it seemed

to us that a five tube method for the determination of the pepsin number would yield results of just as much clinical value as the finer graded seven or ten tube procedure of Givens[2] and of Rose.[1] On this account the following technic was adopted.

Solutions Required

1. Edestin solution: 0.1 gm. of the dry, finely ground edestin is weighed out on a good balance, and triturated with 10 gms. of common salt in a glass mortar, at first carefully and then more vigorously. At first, the edestin tends to collect above the salt on the sides of the mortar, but it is soon scoured off by the salt. As soon as the mixture is uniform, it is poured into a 100 c.c. volumetric flask (or 100 c.c. graduated cylinder), the last particles are brushed in and water is added to the mark. The flask is then placed in a water-bath at 50° C. and shaken frequently until all the salt and protein are in solution. A slight cloudiness, due largely to impurities in the salt, remains and is removed by filtering through a dry folded filter into a dry flask. By adding a little toluol, the solution has been kept by us for seven months without deterioration.

2. Hydrochloric Acid, 0.6 per cent: This is easily made by diluting 16.46 c.c. of normal HCl to 100 c.c. with water.

3. Diluted Gastric Contents (1:20): Five c.c. of the gastric contents to be analyzed, filtered through cotton, are pipetted into a 100 c.c. graduated flask (or 100 c.c. graduated cylinder), diluted to the mark with water, and thoroughly mixed.

Technic of Determination

Using a 1 c.c. pipette graduated in hundredths, 0.85, 0.8, 0.7, 0.5, 0.0 c.c. of water are placed, respectively, in a series of five small test tubes about 1 cm. in diameter. (Ordinary test tubes will do as well.) To each tube is then added 1 c.c. of 0.6 per cent HCl, and 1 c.c. of the 0.1 per cent edestin solution.* The edestin is promptly precipitated out by the acid as a very fine precipitate that soon becomes flocculent. To the first tube, 0.15 c.c. of diluted gastric contents (1:20) is added; to the second tube, 0.2 c.c.; to the third, 0.3 c.c.; to the fourth, 0.5 c.c.; and to the fifth, 1.0 c.c. A sixth tube containing 1 c.c. of the acid, 1 c.c. of the edestin, and 1 c.c. of diluted, *boiled* gastric juice may be added as a control. The total volume is 3 c.c. in each case. The contents are mixed by stoppering the tubes with the finger and inverting, and the tubes are then placed in a water-

TABLE II

SHOWING THE CONTENTS OF EACH TUBE AFTER THE DIGESTION IS SET UP AND ALSO THE PEPTIC NUMBER FOR EACH TUBE

TUBES	1	2	3	4	5
Water, c.c	0.85	0.8	0.7	0.5	0.0
0.6% HCl, c.c.	1.0	1.0	1.0	1.0	1.0
0.1% edestin solution, c.c.	1.0	1.0	1.0	1.0	1.0
Gastric juice, (diluted 1:20) c.c.	0.15	0.2	0.3	0.5	1.0
Peptic number	133	100	67	40	20

*We have used a protein solution of 0.1 per cent as a matter of economy, and because it permits a 1 to 20 dilution of gastric juice, gives a normal peptic number of about 100, and makes less trouble with traces of undigested protein.

bath at 50° to 52° C. for 15 minutes. Table II gives in condensed form the contents of each tube after they are ready for digestion.

At the end of the 15 minute period, the tubes are removed from the bath, the contents are mixed by inverting as before, and the tube is selected which contains the least amount of gastric juice, and in which digestion is complete as indicated by the fact that no precipitate (or only a slight trace) remains. The peptic number, which is taken as an index of the amount of pepsin present, is calculated as the greatest number of cubic centimeters of 0.1 per cent edestin solution that will be cleared up by 1 c.c. of undiluted gastric juice acting for 15 minutes at 50° to 52° C. Hence,

$$\text{Peptic number} = \frac{\text{Dilution of gastric contents} \times \text{c.c. 0.1\% edestin used}}{\text{Smallest number of c.c. of diluted contents causing complete digestion}}$$

But, as 1 c.c. of 0.1 per cent edestin is always used in each tube of the series,

$$\text{Peptic number} = \frac{\text{Dilution of gastric contents}}{\text{Smallest number of c.c. of diluted contents causing complete digestion}}$$

For example, if digestion occurs in Tubes 2, 3, 4, and 5, but not in Tube 1 (cf. Table II), the

$$\text{Peptic number} = \frac{20}{0.2} = 100.$$

In case the gastric contents have been diluted 1:20, the peptic number may be readily obtained by reference to the bottom line in Table II.

TABLE III

SHOWING RESULTS OF DETERMINATIONS MADE BY THE PROPOSED METHOD

CONTENTS		FREE HCL	TOTAL HCL	PEPTIC NUMBER	
Subject	Date			Rose-Givens	Edestin Method
P. R. F.	3/25/18	68 (0.25%)	93 (0.34%)	42	100
P. R. F.	3/26/18	50 (0.2%)	71 (0.26%)	42	100
P. R. F.	3/27/18	60 (0.22%)	82 (0.30%)	42	100
F. H. F.	3/26/18	28	40	42	100
R. C. L.	3/26/18	56	65	31	67
R. C. L.	3/27/18	80	91.5	42	100
G. H.	3/27/18	21	29	18	40
R. C. L.	3/29/18	61	74		67
O. K.	3/30/18	40	56		67
X.	4/ 4/18	—	—	—	100
P. R. F.	11/25/18	—	—	—	67*

*Four determinations of the peptic number were made in this case, using each of the four samples of edestin, respectively (cf. Table I). Identical results were obtained.

The gastric analyses recorded in Table III are normal with a single exception (G. H.) and show that for contents taken one hour after an Ewald test breakfast the normal peptic number by the method proposed is 67 to 100, which happens to be a little more than twice that of the pea globulin method of Givens.[2]

SUMMARY

The method proposed for the preparation of edestin gives a product of sufficient uniformity to be used in the estimation of peptic activity without the necessity of ascertaining its exact edestin content. The procedure for determining peptic activity gives a clearer end point than that of the pea globulin method, less protein is required, and the method, even to the preparation of the edestin, is very practical for clinical use.

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TURKISH WASH CLOTHS FOR PACKS IN EXPERIMENTAL SURGERY*

By F. C. Mann, M.D., Rochester, Minn.

MANY different materials have been used for packs. Chief of these is gauze, and for general use it is doubtful whether any material can be found to replace it. In our own work in experimental surgery, however, we have found that the turkish wash cloth is more satisfactory. These wash cloths have several advantages over other materials which we have used, gauze, rubber, silk and paper. They can be secured easily, various sizes can be obtained, when placed in position they remain in place, and contrary to our preconceived idea, when moist they do not irritate delicate surfaces such as the peritoneum or pleura, nor does any of their rough surface come off into the wound. During more than a year's use they have proved very satisfactory, and they are much cheaper than the other materials.

*Division of Experimental Surgery and Pathology, Mayo Clinic.

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EDITORIALS

The Bacteriologic Examination of Suspected Typhoids

A REPORT from the Public Health Laboratories of Cairo, Egypt, contains a most valuable contribution on the above named subject. Eighteen hundred twenty-seven suspected cases were examined. The report is divided into five parts:

(1) The methods of examination.
(2) The results of the investigation, showing the relative proportion of the cases of typhoid, paratyphoid *A* and paratyphoid *B*.
(3) Conclusions as to the diagnostic value of these bacteriologic examinations.
(4) The characters of the bacilli found.
(5) General conclusions drawn from the investigation.

The methods of examination and the material studied may be divided into the following groups.

I. Blood Cultures.—The method followed in this procedure was a modification of that suggested by Conradi. The hospitals were supplied with test

tubes containing 10 c.c. of sterile ox bile. These were inoculated with from 2 to 5 c.c. of the patient's blood and then sent to the laboratory. The tubes were placed in the incubator where they remained for 48 hours. They were then shaken and a large loopful spread on an Endo-agar plate. After 24 hours in the incubator, the plate was examined. In some instances, 72 hours in the incubator measured the time, but this prolongation seemed to have no advantage.

II. Feces.—No attempt was made to render the stools fluid by the administration of drugs. The material was sent to the laboratory in corked test tubes. If not already sufficiently fluid, a thick suspension in bouillon was made and a large loopful spread on each of two plates of Endo's medium. Other preparations have been tried, but this seems to be as good as any. Both typhoid and paratyphoid bacilli grow well on it. After 24 hours of incubation, the plates were examined, and if suspicious colonies were present, subcultures were made again on Endo's medium with a further incubation of 24 hours. In this way, every culture examined was obtained from a single colony from a culture which had itself originated in a single colony. To subculture from a single colony on the original plate does not guarantee purity.

III. The Urine.—The specimens of urine examined were obtained in the ordinary way without precautions other than the thorough cleansing of the vessel used. Specimens were not centrifuged and two or three drops of each were spread on a plate of Endo's medium. Suspicious colonies appearing after incubation were purified by subculture and examined.

Suspicious colonies were purified by two subcultures before attempting identification. The same procedure was not exactly followed throughout the series. For the greater part of the time, the second subculture was introduced into a bouillon tube and a glucose-bouillon tube, the latter containing litmus and having a small Durham's tube within it to demonstrate the presence of gas. On the third day from the commencement of the examination, the glucose tube was observed, and if it had become acid the bouillon tube was tested with agglutinating sera. Occasionally the growth in the glucose tube showed plainly that the organism was not a member of the typhoid group, revealing perhaps definite turbidity without acid formation or the appearance of a pellicle, when further investigation was unnecessary, but usually the bouillon culture was tested against the three sera,—typhoid and paratyphoid *A* and *B*. Although the presence of gas in the glucose-broth tube may be taken to exclude the typhoid bacillus, its absence after twenty-four hours by no means excludes paratyphoid organisms. All organisms found to be typhoid or paratyphoid were subsequently examined in detail.

During a part of the investigation, cultures of the isolated organism on agar were prepared and tested by means of agglutinating sera. In a few instances in which negative result was obtained, but in which the growth on Endo's medium had been suspicious, the organism was again tested with a positive result. The report states that the method of identification detailed above has resulted in failure to detect any typhoid or paratyphoid bacilli which were not agglutinable by the corresponding serum, but in a routine examination it is necessary to establish a definite criterion on which to base a positive conclusion and

agglutination by the specific serum in a sufficient concentration is probably the most satisfactory that can be adopted.

Of the 1,827 cases examined, positive results were obtained in 297, or 21 per cent. This figure the investigators consider of little significance, either as an indication of the number of cases of typhoid among those presenting the clinical features, or as an expression of the value of bacteriologic examinations in diagnosis. The total number of specimens examined was 5,350, but the number from individual cases varied: from some, seven or more specimens having been received, from others only one. This variable factor may be called the degree of examination, and with this in view they have been divided into seven categories. The results are given in Table I.

TABLE I

GROUP	DEGREE OF EXAMINATION	TOTAL	POSITIVE	PER CENT
I	Six or more specimens, including a blood culture	108	35	32
II	Six or more specimens, but not including a blood culture	68	8	12
III	Four or five specimens, including a blood culture	355	76	21
IV	Four or five specimens, but not including a blood culture	88	14	16
V	Two or three specimens, including a blood culture	292	71	24
VI	Three specimens or less, not including a blood culture	483	42	8
VII	Blood culture only	433	141	32
	Total	1827	387	21

Inasmuch as the cases were examined only for diagnostic purposes, many of those which showed positive findings in the first or second specimen were not further examined. Therefore it is obvious that a certain number of cases in Group V and VII would have been in Group I or III but for this factor. The relative proportion in which the three organisms were found was as follows:

Typhoid ..... 30= 8%
Paratyphoid A .....283=73%
Paratyphoid B .....74=19%

Five cases showing double infection are not included in these figures. One case showed B. paratyphosus *A* from the blood, and B. typhosus from the feces. Two cases showed B. paratyphosus *A* from the blood and B. typhosus from the urine. Two cases showed B. paratyphosus *A* and *B* from the urine.

Beaton, director of the laboratory, speaks as follows concerning the value of bacteriologic examinations as an aid to diagnosis in suspected typhoid:

"Although the only practicable method of arriving at a diagnosis in a suspected typhoid is the agglutination reaction, under certain conditions a bacteriologic examination may be necessary. The diagnosis may be required in the first week of sickness when agglutination is not to be depended upon, or the reaction may be indefinite or fail altogether in a case in which the clinical and other conditions point positively to a typhoid infection, or the agglutination reaction may be complicated and perhaps rendered useless by recent vaccination, or the question may not be one of simple diagnosis but of the continuance of infectivity in a convalescent or the presence of bacilli in a carrier. Under any of these circumstances it becomes necessary to look for the offending bacilli in order to arrive at a definite conclusion, and if the bacillus be found this conclusion is at-

tained, but on the other hand if the bacillus be not found, no definite conclusion can be formulated. While a positive result is, other than under exceptional conditions, conclusive, a negative result is regarded as having no significance, although a negative conclusion may become more and more justifiable as the number of negative results increases. All diagnosis, clinical, bacteriologic, or otherwise, resolves itself ultimately into a question of probability. It has therefore appeared worth while to employ the considerable number of examinations involved in this investigation in an effort to throw some light on the question of the degree of probability of arriving at a positive conclusion from the bacteriologic investigation of a case of typhoid.

The number of cases in which cultures from the blood were made was 1,188, and of these 226 or 19 per cent were positive. Table II shows the time in the progress of the disease when blood cultures were made.

TABLE II

BLOOD CULTURES

	TOTAL	POSITIVE	PER CENT
All cases	1,188	226	19
First week	199	65	33
Second week	355	60	17
Third week	145	13	9
Later than third week	118	8	7
Unknown	371	80	—

These figures show that the probability of isolating the causative organism from the blood in the second week is only half that in the first week, and in the third week again only half that in the second week. However, it is seen that even in the fourth week a small percentage gave positive results.

In 206 cases shown by finding the bacilli in the blood or the urine to be actually some form of typhoid fever, the bacilli were found in the feces in only 12 per cent. If two or three specimens of feces were examined, one of which proved positive, it was only exceptionally that the other one or two proved positive too, and in the event of more than three specimens being examined, in no case did all the specimens prove positive. In 46 cases in which two or more specimens were examined and in which at least one specimen proved positive, in only eight did more than one specimen prove positive.

In 297 cases in which typhoid bacilli were found in the blood or in the feces, the same organism was found in the urine in only 6 per cent. In 51 cases in which two or more specimens of urine were examined, and in which at least one specimen proved positive, in only nine did more than one specimen prove positive.

The characters of the typhoid and paratyphoid bacilli obtained were briefly studied. In addition to the specific agglutination test, the action of each organism on carbohydrates and on milk was tested. The Bacillus typhosus should produce no change in lactose, saccharose, and dulcite, and should form acid but no gas in mannite and glucose. Of 30 strains examined, 29 gave this typical reaction, although in one case the amount of acid formed from mannite was very small. In the case of one strain, a bacillus isolated from the urine, no acid was formed from mannite after five days; this strain was agglutinated by a typhoid serum at a dilution of one in 1600.

The Bacillus typhosus consisting of 13 strains was tested on litmus milk. All produced a decided acid reaction within 24 hours and this persisted for more than a week in every case. At the end of the second week with a certain number of the strains the medium had lost its acidity, and by the end of the third week a diminution of acidity had occurred in every case; 5 strains showed reduced acidity, 6 had returned to the original color of the medium, and 2 had become definitely alkaline. No clotting occurred.

The action of Bacillus paratyphoid *A* on carbohydrates is characterized by the formation of gas and acid in dulcite, mannite, and glucose, while no change occurs in lactose and saccharose. Dr. Beaton states:

"The strains of B. paratyphosus *A* isolated proved to be very erratic in their action, and to present in this respect a striking contrast to the strains of B. typhosus and of B. paratyphoid *B*. While conforming to type in their action on lactose and saccharose (although it must be admitted that a definitely lactose-fermenting organism would have escaped recognition) they presented a considerable amount of variety in their action on dulcite, mannite, and glucose. Of 303 strains examined, 238 gave the typical reaction to some degree, although only 94 produced a sufficient quantity of acid and gas for the reaction to be considered wholly typical. The two strains giving acid only in mannite and glucose, the typical reaction of B. typhosus, were agglutinated by a paratyphoid *A* serum at a dilution of 1 in 500, and were not agglutinated by a typhoid serum."

Two hundred strains of this organism were studied as to their action on litmus milk. All produced acid after three days, without the formation of a clot, and all remained acid after thirty days, still without clotting. This constitutes the typical reaction.

The characteristic action of Bacillus paratyphoid *B* on carbohydrates consists in the absence of change in lactose and saccharose and the formation of acid and gas in dulcite, mannite, and glucose. Of 78 strains examined, all gave the typical reaction. In two, the quantity of gas formed from dulcite and glucose was small.

Twenty-four strains of Bacillus paratyphosus *B* were examined as to their action on litmus milk. All produced acid during the first 24 hours, and this reaction persisted on the second day. On the third day, more than half the strains showed a definite alkalinity, and at the end of the first week, with 20 strains, the medium was definitely alkaline. After three weeks all strains showed a strongly alkaline reaction.

The most important conclusion to be drawn from the data is the relative proportion of cases of typhoid and paratyphoid *A* and *B* found among typhoids acquired in Egypt and those acquired on the Gallipoli Peninsula. Among those from the Peninsula, 82 per cent showed paratyphoid *A*, and 18 per cent paratyphoid *B*. There were among these no typhoids. Among 40 cases known to have originated in Egypt, 5 per cent were typhoid; 87.5 per cent, paratyphoid *A;* and 7.5 per cent, paratyphoid *B*. In many of the cases it was uncertain where the disease originated. The figures show at any rate a great preponderance of paratyphoids in both groups. No definite information is available as to the amount of paratyphoid fever in Egypt before the war, but it may be said that paratyphoid *B* occurred, although to a much smaller extent than typhoid; while

cases of paratyphoid *A*, although probably occurring occasionally, were very rare. These conclusions are drawn entirely from the results of agglutination tests, and it may be assumed for all practical purposes that paratyphoid *A* was not endemic in Egypt.

It is too early to determine whether paratyphoid *A* will be established as one of the endemic diseases of Egypt, but this should in due course constitute an interesting point in connection with our knowledge of the factors determining the existence of endemic diseases, and a curve based on the relative proportion of cases of typhoid and paratyphoid *A* and paratyphoid *B* occuring in Egypt during the next few years should throw some light on the question of how far the absence of this disease from a geographical area may be due to the fact that it has never been adequately introduced into the area, and how far to the lack of conditions suitable for its development.

This report is most interesting, and at the same time, highly suggestive. It will be observed that by all the tests combined only 21 per cent proved to be one of the known forms of typhoid fever. It would be highly interesting to know what the final decision was in the remaining cases. If there be three typhoid fevers, etiologically considered, why should there not be more? If there be organisms which we now designate as paratyphosus *A* and *B*, there may be varieties which we will later designate as *C* and *D*, and so on. It is possible and indeed highly probable that so far as vaccination has failed it is due to the disease being caused by other members of the typhoid group, which in all probability is much larger than we now appreciate.

A second most interesting point brought out in this valuable report is that of the introduction of a disease into a country where it has not hitherto prevailed, and the possibility of the newly introduced organism supplanting kindred organisms already native to the country. There are many reasons for believing that in the various camps in this country different organisms became predominant and the dominating organism changed from time to time with new importations. It is possible that the great differences in the death rates in the various camps in this country may have been due to this or some similar cause.

—*V. C. V.*

The Scientific Research Fund for Study of Venereal Diseases

THE United States Interdepartment Social Hygiene Board, through its executive Secretary, T. A. Storey, Washington, D. C., announces appropriations from its Scientific Research Fund to the following institutions for the purpose of investigating "more effective medical measures in the prevention and treatment of venereal diseases:"

University of Nebraska College of Medicine

1. (a) "Investigation relative to the development of an internal urinary antiseptic."

(b) "Investigation of the value of certain anilin dyes in the treatment of gonorrhea." Both under the direction of E. G. Davis, M.D., Director of Pathological Laboratory.

St. Louis University College of Medicine

2. "Studies in infection by gonococci." Under the direction of R. A. Kinsella, M.D., Director of Department of Experimental Medicine.

Woman's Medical College of Pennsylvania

3. "A serological study of syphilis in pregnant women and new-born children with special reference to the efficacy of the accepted methods of syphilitic treatment." Under the direction of Berta M. Meine, M.D., Director of Research Department.

Washington University School of Medicine

4. "The laboratory (biological) investigation of the latent syphilitic as a 'carrier.'" Under the direction of Martin F. Engman, M.D., Professor of Dermatology.

Cornell University Medical College

5. "Serological study of the gonococcus group." Under the direction of John C. Torrey, Ph.D., Professor of Hygiene.

Jefferson Medical College of Philadelphia

6. "A series of studies for the recognition and diagnosis of treponema pallidum in venereal diseases, and the effect of various drugs and materials as germicidal agents against treponema pallidum." Under the direction of Randle C. Rosenberger, M.D., Professor of Hygiene and Bacteriology.

Yale University Medical School

7. (a) "Effect of anilin dyes, particularly Gentian Violet on the gonococcus with reference to the growth of the organism in media containing the dye and with reference to the effect on organisms in tissue (therapeutic effect)." Under the direction of J. M. Flint, M. D., and J. W. Churchman, M.D., Professors of Surgery.

(b) "An intensive study of methods for the isolation and identification of the gonococcus with a view to the determination of the homogeneity or heterogeneity of strains and their etiological relationships." Under the direction of George H. Smith, M.D., Assistant Professor, Department of Pathology and Bacteriology.

(c) "The demonstration of syphilitic nature of unusual lesions encountered at the post mortem table." Under the direction of M. C. Winternitz, M.D., Professor of Pathology and Bacteriology.

Union University Medical Department, Albany

8. "For studies on the nature of the Wassermann reaction." Under the direction of Thomas Ordway, M.D., Dean, and Associate Professor of Medicine.

The United States Interdepartmental Social Hygiene Board is composed of—

Carter Glass, Secretary of the Treasury,
Newton D. Baker, Secretary of War,
Josephus Daniels, Secretary of the Navy,
Lt. Col. W. F. Snow, Medical Corps, U. S. A.,
Lt. Comdr. J. R. Phelps, Medical Corps, U. S. N.,
Asst. Surg. Gen. C. C. Pierce, U. S. Public Health Service,
Thomas A. Storey, Executive Secretary.

Up to June 21, the Board had approved appropriations from its Scientific Fund for twenty researches distributed among twelve medical schools.